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ESPECIALLY PREPARED ORIGINAL ARTICLES

ON

TREATMENT, MEDICINE, SURGERY, NEUROLOGY, PEDIATRICS, OBSTETRICS, GYNECOLOGY, ORTHOPEDICS, PATHOLOGY, DERMATOLOGY, OPHTHALMOLOGY, OTOLOGY, RHINOLOGY, LARYNGOLOGY, HYGIENE, AND OTHER TOPICS OF INTEREST TO STUDENTS AND PRACTITIONERS

BY LEADING MEMBERS OF THE MEDICAL PROFESSION THROUGHOUT THE WORLD

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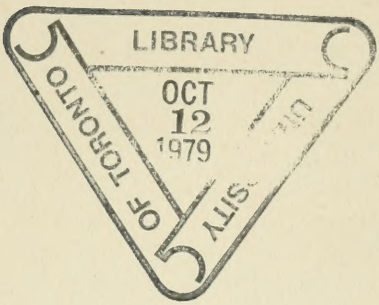

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Treatment

THE PROGNOSIS AND TREATMENT OF THE CHRONIC VALVULAR DISEASES OF THE HEART

BY DELANCEY ROCHESTER, M.D.

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The factors to be taken into consideration in the prognosis of chronic valvular diseases of the heart are: (1) The valve affected; (2) the nature of the lesion; (3) its effect upon the myocardium; and (4) the resultant effect upon the capillary circulation in general and upon the pulmonary and the portal circulations in particular. Other factors of some value in the prognosis are the main etiologic factor concerned in the production of the especial valvular lesion under consideration, and the associated conditions of the arterial walls.

So far as the underlying etiologic factor is concerned in any given case of chronic valvular deformity, the prognosis is better if this is one of the acute infections, such as scarlatina, rheumatism, etc.; the outlook is not so good if it is a chronic infection, or a degenerative process of the valve itself or of the myocardium. The reason for this is plain. If the acute infection has not destroyed the individual and the recovery has been complete, there is merely the valvular deformity present and nothing to interfere with the development of complete hypertrophy of that portion of the myocardium which will compensate for the valvular defect. On the other hand, if there is a chronic infection, such for example as syphilis, it is continuously active, not only upon the valve but upon the myocardium and upon the general nutritive processes of the body; if it is a degenerative lesion that produces the valvular defect, the degenerative process is as fully advanced in the rest of the circu-

latory apparatus, especially the myocardium and the arterioles, and an establishment of good compensation or the reëstablishment of broken compensation is, in all probability, an impossibility. Thus the age of the patient at the time the lesion is acquired has a decided bearing on the prognosis. In a general way we may say that after five years of age the younger the patient when the disease develops the better the outlook, provided that at the time the patient is kept quiet for a long enough period, that nutrition is well maintained, and that when the proper time comes exercise is undertaken only under competent supervision.

In any valvular lesion acquired after middle life, the prognosis is not so favorable even if the result of an acute endocarditis, because even though the valvular lesion may remain stationary, the myocardium is unable to hypertrophy sufficiently to compensate for the defect. An exception to these general statements must be made of mitral stenosis, which is usually inflammatory in origin and usually first arises in early life, but is steadily progressive in character, and thus continually increases the obstruction to the onflow of the blood.

In this discussion in order to keep within reasonable limit, I shall omit reference to primary defects of the valves of the right side of the heart. Of the single valvular lesions of the left side the most serious, in my opinion, is aortic insufficiency. True aortic stenosis and true mitral stenosis are, I think, of equally serious import and stand closely second to aortic insufficiency. Simple mitral insufficiency I consider the least serious of the several single valvular lesions of the left side of the heart.

While these general considerations remain true we must never lose sight of the fact that failing compensation in any valvular lesion makes it a most serious affection, and that there is a much worse outlook for the future in a case of mitral insufficiency in which the right ventricle is dilated than in an aortic insufficiency in which there is sufficient compensatory hypertrophy of the left ventricle. Combinations of two or more valvular defects, even though one may be a so-called safety-valve leak, are more serious than any single defect.

It is worth while for us to consider briefly the mechanism of the circulation when the valves are defective, the means that nature

uses to overcome the defect, and the result of the failure of nature to establish compensation, that we may the more readily recognize clinically the beginning of the failure and take the proper steps to arrest the progress and reëstablish compensation.

Aortic Insufficiency.—In aortic insufficiency during ventricular systole the blood is driven out of the ventricle into the aorta: this is immediately followed by diastole, during which the ventricle normally receives its full quota of blood from the auricle, but, with an insufficient aortic valve, receives also a considerable reflux from the aorta. As a consequence the ventricle is distended beyond its normal capacity and we have an enlargement of its cavity; at the same time nature, responding to the call, increases the number and size of the muscle fibers of the ventricle to such a degree that the heart is able to throw this increased bulk of blood into the aorta and so, even though part of it returns with each diastole, the circulation is kept up, nutrition is maintained, and we say that compensation from the leak is complete. So long as compensation is perfectly maintained the patient complains of nothing, excepting perhaps the feeling of the stronger impulses of the hypertrophied heart during the systole. The evidences of aortic insufficiency are the Corrigan water-hammer pulse, the heaving impulse, the diastolic thrill, usually felt with greatest intensity along the left border of the sternum, the increased area of dulness, chiefly to the left and downward, the prolonged and intense first sound of decidedly booming quality, and the diastolic murmur accompanying the second sound, heard with greatest intensity at the junction of the third right costal cartilage and sternum, transmitted downward along the left edge of the sternum. It is also occasionally heard at the apex,—presystolic in time, the so-called Flint murmur. The reason why this may be considered the most serious of the valvular defects is because on account of the continued reflux of blood into the ventricle and the consequent tendency to great over-distention of this cavity it is with the greatest difficulty that compensation is maintained. Thus conditions which, under normal circulation would be but slight or temporary disturbances to metabolism, are apt to become, with this lesion present, enough to turn the balance in the wrong direction and start the train of events which, unless stayed, lead to dissolution. The picture of broken compensation

with congestion and edema of the lungs, engorgement of the liver, anasarca, ascites, hydrothorax, and the accompanying symptoms of cyanosis, dyspnea, anorexia, dyspepsia, impaired cerebration, impaired locomotion, precordial distress, etc., it is not necessary to dwell upon, since it comes eventually when the failure is complete, and when treatment is little more than an attempt to render the unfortunate subject as comfortable as possible for his remaining days. What I wish to call attention to is the evidence of beginning loss of compensation and the measures to be adopted to reëstablish the compensation.

In aortic insufficiency the first evidences of failing compensation usually show themselves in the physical signs before they are far enough advanced to produce subjective symptoms. It is important, therefore, if one has the care of a case of aortic insufficiency to impress upon the patient or some member of the family the necessity of being examined at stated intervals.

When the lesion of the aortic valve is slight or moderate and compensation is complete we find only a moderate diastolic collapse of the pulse, and while the murmur usually replaces the aortic second sound in the aortic area and below it, we can usually hear the sound at the sternoclavicular junction and over the right carotid. In a case in which it has been possible to hear the second aortic sound in these regions, one of the earliest evidences of beginning failure of compensation is the weakening or disappearance of this sound. Coincident with this the collapsing character of the pulse at the wrist and in the carotids becomes more marked and very soon the capillary filling and emptying may be demonstrated under the finger-nails or in the lip. Associated with these is an increasing tendency to shortness of breath and palpitation of the heart on exertion. The next sign is a shortening and lessening in intensity of the first sound at the apex, the short silence becoming longer so that the sounds are about equally spaced and approach each other in the pitch and quality as well as in their length. Up to this point, provided there is not serious degeneration of the arteries or Bright's disease or other chronic disease, I am of the opinion that restoration to the previous condition of a well-conducted circulation is possible, but if the dilatation goes a step further and we have the yielding of the mitral valve, showing that the dilatation has included

the auricle and auriculoventricular orifice,—as evidenced by the development of an apical systolic murmur transmitted to the axilla,—then I believe that while by judicious management we may for a time slow the progress of the disease, it cannot be stayed, much less can the circulation be restored to its previous state. This state of affairs is indicated by the evidence of pulmonary congestion, venous pulsation, engorgement of the liver and portal circulation, dropsies, etc.

In aortic insufficiency we must remember that we have a lesion of the heart that renders the patient susceptible to sudden death more often than any other form of valvular disease,—that any sudden physical exertion or any prolonged and fatiguing physical or mental exertion or any sudden emotion, whether of grief, pleasure or joy, may so affect the peripheral circulation as suddenly to increase the reflux of blood into the ventricle to such a degree as to prevent the muscular fibers from contracting and to stop the heart in diastole.

Aside from the physical signs, the pulse, and the several dropsies referred to, symptoms of serious import are often connected with the brain, probably due to insufficient blood supply. Such symptoms are failing memory, inability to concentrate thought, mild hallucinations of temporary but recurring character, and occasionally pronounced delirium, usually associated with insomnia and restlessness.

"Perhaps more can be done," says Broadbent, "to prolong life and postpone suffering in aortic regurgitation than in any other form of valvular disease: at any rate, it is in this disease that the greatest difference can be made by care on the one hand and imprudence on the other. A patient may die suddenly from a simple rash act who might have lived twenty years, or condemn himself by a single imprudence to a short and suffering existence, when fair health and many years of life were possible for him." Rest, complete and absolute, in bed, is the most important therapeutic measure in this form of disease. The period of rest should be as long as is necessary to accomplish the desired end, whether that rest be two weeks or six or eight months. After this period of rest exercise may be begun gradually, but with the aid of a competent masseur, assisted and passive at first, then resisted and finally active, walking

a definite distance each day, the distance to be gradually increased until a walk of a mile or two is taken without fatigue. Then the rate of walking may be gradually and very carefully increased, the pulse and the heart's action being carefully studied and recorded before and after the exercise. The possibility of sudden death makes it important that these patients should be warned against over exertion of any sort, physical or mental. "Go slow" should be their motto.

As already indicated, death may occur in this form of disease either from failure of the left ventricle to fill the capillaries or from back pressure into the veins through right ventricular failure secondary to that of left. The treatment of the two conditions is quite different. It is important, therefore, to recognize the condition present. In the first, the symptoms are more apt to be central,—insomnia as described, or a distressing dyspnea of an expiratory type,—accompanied by marked pallor and an anxious, suffering expression of countenance. Dropsy, if present at all, is very slight and is apt to be limited to a little edema at the base of the lung or a little fluid in one or both pleural cavities. In this condition nutrition, stimulation, and oxygen are especially indicated. Drugs that are useful in such cases are nux vomica or strychnin, aromatic spirit of ammonia, compound spirit of ether, and belladonna or atropin. In case there is arteriosclerosis present also, nitroglycerin is especially useful. A combination which I have used in these cases with good effect is the powdered extract of nux vomica, spartein sulphate, and alcoholic extract of belladonna, and the nitroglycerin when indicated. In all forms of aortic valve disease we are very apt to have attacks of angina pectoris, and amyl nitrite or nitroglycerin gives the best results. For the sleeplessness and restlessness there is nothing better than morphin hypodermatically. Sometimes when it disagrees or seems to produce constipation we get good results by the use of paraldehyde in full dose. It is remarkable what a night or two of good rest will do for these cases.

Iron, arsenic, and phosphorus all have strong advocates for use in this and other forms of heart disease when the nutrition of the myocardium is failing, and each or combination of all may be used with benefit. When the disease has gone further and we have the right ventricular failure our line of treatment is decidedly different.

Here we have in fact a mitral insufficiency added to our aortic disease and we have to relieve the dropsies that are accumulating, in order to give the heart any sort of a chance. Under such circumstances a mercurial purge is of especial value, or we may use for 24 or 48 hours the Niemeyer pill of blue mass, squill, and digitalis, given every 4 hours and followed by Epsom salt. In the use of digitalis in aortic regurgitation, however, we must be very careful, and as soon as the dropsical symptoms have disappeared it should be stopped. The reasons for not using digitalis in this form of valvular defect are apparent if we consider the mode of action of digitalis. It is of value in that it causes a forceful contraction of the ventricle during systole and tends thus to help the ventricle empty itself, but we must remember that at the same time it causes a contraction of the systemic arterioles, thus increasing the pressure against the ventricle and making it hard to empty itself; and at the same time it slows the diastole of the ventricle, thus giving a longer period for the ventricle to receive the blood from the auricle and that regurgitating from the aorta, thus tending further to stretch the incompetent ventricle muscle and produce greater dilatation. Other purgatives and diuretics should be used, and in some cases great benefit is secured by the use of a hot-air sweat in a cabinet or in bed.

Aortic Stenosis.—True aortic stenosis is a relatively rare disease, autopsies proving that in many cases in which a loud systolic murmur is heard in the aortic area and transmitted to the vessels of the neck, there is no true stenosis, but merely a roughening of the orifice or valves, sufficient to throw the stream of blood into such currents and eddies as to produce a murmur, but not to produce any true obstruction to its escape from the ventricle. To determine that there is true stenosis we must have, besides the systolic murmur, a moderate hypertrophy almost entirely confined to the left ventricle, and the pulse characteristic of the lesion,—namely, a pulse usually of moderate volume which rises slowly, persists at its maximum for some time and falls slowly, the vessel being generally small and full during the diastole and of moderate tension approaching high. Without such pulse, even with the murmur and hypertrophy, we cannot say positively that we have aortic stenosis.

Aortic stenosis, once established, has a tendency slowly but stead-

ily to increase, so that in order to compensate for it there must be an increasing hypertrophy of the left ventricle. So long as this hypertrophy keeps pace with the stenosis and fully compensates for it, there will be no subjective symptoms. There comes a time, however, when, either through physical or mental strain, intercurrent disease or advancing years, the nutrition of the myocardium is diminished and the compensation fails, the ventricle is no longer able to empty itself completely, there is not room for all the blood from the auricle, the ventricle dilates, the mitral orifice is stretched, a leak occurs, pulmonary congestion and edema result, and if the patient lasts long enough right heart failure ensues and general dropsy follows in its train.

Sometimes the aortic stenosis is so marked that the contraction of the powerfully hypertrophied left ventricle puts such a strain on the leaflets of the mitral valve and upon their chordæ tendineæ that they yield before the pressure and mitral insufficiency occurs. Thus we see that in either case the development of mitral insufficiency in a case of aortic stenosis is a matter of bad prognostic import. Sudden death in cases of aortic stenosis is rare unless the disease has associated with it angina pectoris and arteriosclerosis. Nevertheless, few subjects of aortic stenosis live to be sixty years of age, most of them dying under forty-five years.

The symptoms and signs which may be considered as indicating failing compensation in these cases are briefly as follows: A tendency to shortness of breath on exertion, accompanied by precordial distress and sometimes by true angina pectoris; a loss in tension of the pulse; weakening or disappearance of the aortic second sound; weakening of the first sound at the apex; and the development of a mitral systolic murmur. Following this, right ventricular failure and all the resulting venous obstructive dropsies, etc., are likely to develop.

When such failure is distinctly due to some over-exertion or other definite single cause which may be avoided, the previous state of health may sometimes be reëstablished by prolonged rest in bed, a carefully selected diet, and the very gradual resumption of exercise. Under these circumstances digitalis, strophanthus, or cactus, either alone or combined with nux vomica and belladonna, will be of decided value.

When, however, these symptoms have developed gradually and insidiously and cannot be traced to any single cause, it means that the resisting power of the individual and his capacity to develop myocardial strength have reached their limit, and little can be done to improve materially the patient's condition or postpone the end. On this account it is of utmost importance that when we have a patient with aortic stenosis under our care we should see to it that his general health is kept up to normal, that his excretions are well carried on, and that his blood does not fall below par.

When with either of the aortic lesions there is associated arteriosclerosis, or chronic nephritis, as there very frequently is, the outlook is so much the worse. In many such cases the arteriosclerosis is especially advanced in the aorta and the coronary arteries. Under such circumstances angina pectoris is commonly present and makes the prognosis more serious. Amyl nitrite inhalations generally give the most prompt relief, but not infrequently have to be reinforced by morphin hypodermatically. Atropin with the morphin has a good effect upon the heart and should always be used with the first injection, taking care not to repeat it too often. The combination of aortic stenosis and aortic insufficiency is not very common, though the occurrence of a double murmur at the aortic orifice is not at all infrequent. It usually means an aortic insufficiency and a roughened valve. Nevertheless the combination does sometimes occur, and occasionally the supervention of a stenosis upon a previously-existing insufficiency has had a conservative effect when there has been sufficient reserve power in the individual to develop a compensating hypertrophy, the constriction of the orifice tending to lessen the amount of regurgitation during diastole and so to diminish the tendency to dilate. Usually, however, the double lesion merely puts together the evil effects of both and hastens the inevitable end.

Before leaving the consideration of the aortic orifice, I wish to make plain that I think a great deal of harm has been done by the indiscriminate use of digitalis in treating these lesions; that its use is decidedly contraindicated in aortic insufficiency excepting for short periods of time when compensation has failed to that degree that the mitral orifice has dilated and we have right heart failure and its concomitants of venous obstruction. Then, even, digitalis

should not be used alone, but always associated with some other more powerful eliminant, and the digitalis should be stopped as soon as the indications for its use have disappeared.

In aortic stenosis digitalis is more useful and it can be used with benefit over a longer period of time, but it has its best results when combined with nitroglycerin, nitrite of amyl, or small doses of sodium iodide or the regular use of the hot-air bath, all of these therapeutic agents causing some dilatation of capillaries, and thus offsetting the danger of arteriole contraction and increase of pressure against the ventricle which digitalis produces.

Mitral Stenosis.—Next to aortic insufficiency, mitral stenosis is the most serious valvular defect of the left side of the heart. It is rarely that persons suffering with this disease reach their fortieth year. It occurs very much more frequently in women than in men, and is usually of inflammatory rather than of degenerative origin. Moreover it is almost invariably progressive, steadily becoming worse, in this respect differing from the other defects of inflammatory origin. In order to formulate a prognosis we must be able to recognize not only the presence of the stenosis, but the degree to which it has progressed. Therefore, we shall have to review briefly the anatomic and functional derangements resulting from the obstruction, and the changes in symptoms and in physical signs that accompany the progress of the lesion.

The first result of the obstruction is a dilatation and hypertrophy of the left auricle. As the disease progresses, however, this is not sufficient to prevent the pulmonary venous stasis and there develops a considerable hypertrophy of the right ventricle. This hypertrophy of the right ventricle, with little or no hypertrophy of the left ventricle, is the most marked result of mitral stenosis and gives rise to some of its distinctive signs. The very great tendency to pulmonary congestion and the rapidity with which this may at times develop is another of the striking phenomena associated with this disease. On account of the collection of a large amount of blood in the left auricle there is considerable tendency to the deposition of fibrin in the auricle and upon the edges of the valve, and embolism and infarction are not uncommon accompaniments of mitral stenosis, the brain being the most common seat of such infarction.

As the disease progresses, the right ventricle has more and more

work thrown upon it, and it is in this disease especially that we are apt to have the so-called safety valve insufficiency of the tricuspid develop. Following this, congestion and not infrequently pulsation of the liver occurs. It is seldom that the patient lives for the development of the dropsies that we might naturally expect to ensue, though not infrequently a certain amount of ascites will be found if looked for. In the first stage of the disease when the hypertrophy of the left auricle is sufficient there usually are no symptoms, and unless the lesion has developed under observation, in the course of an acute infection, a physician is not consulted. In this stage the physical signs are confined to palpation and auscultation, inspection and percussion revealing no abnormality. Upon palpation there is found the presystolic thrill very limited in area, just to the right and a little above the apex, undoubtedly produced by the forceful contraction of the auricle in the attempt completely to empty itself through the constricted mitral orifice. The same thing may be recognized as a presystolic murmur by auscultation. This period of its existence, as I have said, rarely comes before the physician and, even if it does, it requires no treatment beyond the general warning to go slow in all things. The next stage, where there is hypertrophy of the right ventricle, is ordinarily the first to cause subjective symptoms, and is commonly called the first stage of the disease. The symptoms usually presented are shortness of breath on exertion, a tendency to vertigo or light headedness, and epigastric pulsation. This last is often a very disturbing symptom, and is not infrequently accompanied by disturbance of gastric digestion.

In this stage the hypertrophy of the right ventricle is commonly so great that it over-rides, as it were, the left ventricle so that no part of the left ventricle comes to the front of the chest to form the apex. We must bear this in mind when considering the physical signs.

Upon inspection we note the epigastric pulsation. Upon palpation we find that the pulsation in the epigastrium and especially in the precordium just to the left of the ensiform cartilage is powerful, while at the apex it is less strong. We still note the presystolic thrill. Upon percussion we note the increase in area of dulness, chiefly to the left but not much below the normal level. Upon auscultation, besides the presystolic murmur we note the relative

weakness of both the first and second sounds at the apex, the greater intensity of the first sound just to the left of the left sternal line and occasionally over the lower portion of the sternum, and the marked accentuation of the pulmonic second sound at the base as compared to the aortic.

In this stage we have entered upon the danger period in mitral stenosis. A little carelessness in mode of life, a pronounced chilling of the surface of the body, an overloading of the stomach, the occurrence of constipation and straining at stool, an attempt to lift a weight or run for a car or hurry upstairs, a sudden or severe mental or emotional shock, whether of pleasure or pain, might any of them produce so severe a strain upon the heart that, if the result were not immediately fatal, the effect upon the myocardium would be so bad that weeks or months might be required to restore anything like the equilibrium which previously existed.

As we approach the last stage in which the right ventricular hypertrophy fails to compensate, we have an increase in the serious symptoms and pronounced changes in the physical signs. Cardiac palpitation and precordial distress, alternating constipation and diarrhea, anorexia, cough with bloody or blood-stained expectoration, enlargement of liver, ascites, diminution of urine high in color, strongly acid, containing a large amount of indican and usually some albumin and casts are among these symptoms. The pulse of mitral stenosis is generally small and of low tension. In this stage it is generally markedly irregular both as to the time, size, and tension of the beats, the tension usually being decidedly low.

The physical signs of this stage are, upon inspection, a tendency to cyanosis or bluish pallor, and an irregular pulsation in the precordium and epigastrium; upon palpation the thrill is just as apt to be felt early in diastole as in presystole; sometimes it is possible to distinguish two distinct thrills. With the advancing failure the presystolic thrill usually disappears. The cardiac impulse is markedly uneven,—an occasional pronounced impulse and several weak and irregular ones. Percussion shows increase in the area of cardiac dulness usually in all directions, but never so great as in aortic insufficiency. Auscultation shows weakness of the first sound and the systolic murmur of the tricuspid leak, the early diastolic murmur and the disappearing presystolic, and accentuation of the

pulmonary second sound. Sometimes the tricuspid murmur can be heard over the liver, which is always increased in size in this stage of the disease. Rarely the liver may be felt to pulsate. When the disease reaches this stage permanent improvement is out of the question, and a restoration to a state of moderate comfort with a very quiet life is the best that can be expected. A patient in this condition with pulsating veins, enlarged liver, cyanosis, dyspnea and dropsies of various kinds can sometimes be relieved of this serious state in which the left auricle and right heart are distended, by the prompt opening of a vein and the withdrawal of 200 to 400 c.c. of blood. This withdrawal gives of course only a temporary relief and cannot be repeated too often, but I am sure it has occasionally saved a life and started the patient on several months of comfort.

At the same time, or if the demand for relief is not so pressing, leeches may be placed over the precordium or the hepatic region, and a full dose of mercury administered, to be followed by concentrated solution of salts.

In this lesion it appears to me that digitalis and its congeners are especially indicated, and I am sure that I have seen good results from the continuance of the drug over a long period of time. Digitalis slows the diastole and so gives the auricle a longer time to empty itself and increases the force of the systole both of auricle and ventricle, thus giving greater strength to the left auricle in its attempt to empty itself, and to the right ventricle in its efforts to aid the left auricle. Its only possible evil is its constriction of the systemic capillaries, and that can do little harm in this particular case, as the tendency is rather toward a softness of the arterial system.

Mitral insufficiency, the most common and usually the least serious form of valvular disease, next claims our attention. Mitral insufficiency may be of several kinds,—that is, it may be due to deformity of the valve, congenital or acquired; it may be due to deficient strength of the musculi papillares, to which the chordæ tendineæ are attached; it may be due to disease of the chordæ tendineæ; it may be due to dilatation of the auriculo-ventricular orifice.

Sometimes two or more of these conditions are active in the same case at one time. As a result of mitral incompetence we have dila-

tation and hypertrophy of the left auricle and resulting hypertrophy of the right ventricle. From the high pressure consequently maintained in the left auricle, the blood rushes in large amount and with considerable force into the left ventricle during diastole, distending it and consequently producing dilatation and hypertrophy of the left ventricle,—both ventricles thus entering into the enlargement of the heart which occurs with this lesion and is sometimes exceedingly great.

If the left auricular hypertrophy and the right ventricular hypertrophy are sufficient to maintain the blood-pressure to that degree that the systemic circulation is well carried on, there are no subjective symptoms of this disease. The murmur of mitral insufficiency is systolic in time, accompanying or taking the place of the first sound. It is heard usually with greatest intensity at the apex and is heard also to the left of the apex, even into the axilla; it is also heard behind in the lower part of the interscapular region; it is sometimes heard with greatest intensity in the second and third intercostal spaces anteriorly, just to the left of the pulmonary area. Prognostically, so far as the characteristics of the murmur are concerned, it may be said that if the murmur is loud and long it is of less serious import than one which is weak and short. The character of the murmur, however, is not of very great prognostic import except as it changes,—a murmur which has been loud, becoming faint and short usually means that the muscular wall is becoming so weak as not to propel the blood with sufficient force to produce a murmur. There are, however, exceptions to this, too. A murmur which entirely takes the place of the first sound is of more serious import than one which accompanies the first sound.

If the heart is not materially enlarged, and the sounds at the base balance fairly well, the pulmonic being but slightly accentuated, no matter how loud the murmur may be or what is characteristics, the outlook is not bad. The evidences of failing compensation in mitral insufficiency are those of pulmonary congestion and edema, and of right heart failure and general venous engorgement and dropsies. The picture is familiar to all. It is remarkable how many patients with mitral insufficiency will seem to be at the point of death and will so far recover as to go about their business again for weeks, months, or years before another attack comes. I

have been mistaken so many times as to the outcome in such cases that I now never give a bad prognosis in a case of mitral insufficiency, no matter how great the dilatation nor how marked the dropsy, until I have had the patient under observation sufficiently long to know what can be done. The condition of the peripheral circulation, the degree of tension in the general arterial system, is in my opinion the most important factor in making a prognosis in a given case of mitral insufficiency. The amount of peripheral resistance that must be overcome to carry on the circulation is the index of our prognosis. The greater the resistance the worse the prognosis. In a given case of mitral insufficiency in which compensation has failed, of course the two most important things to do are to put the patient as completely at rest as is possible and to relieve the peripheral tension by opening up the emunctories by a mercurial and saline, by diuretics (if the kidney is not too far diseased), and by inducing sweating by means of hot-air or steam preceded by a hypodermic of pilocarpin hydrochlorate, at the same time holding up the heart by the use of nux vomica and digitalis or caffein. Fluid extract of apocynum cannabinum, either alone or associated with fluid extract of digitalis, often has a most salutary effect in getting rid of the edema by the bowels and the kidney. The Niemeyer pill of digitalis, squill, and blue mass or calomel is often of the greatest service in these cases.

Combinations of two or more lesions of the valves are more serious than any of the single lesions with the one exception, already referred to, where an aortic stenosis develops after an aortic insufficiency while the individual is still young and vigorous enough to develop compensatory muscle-tissue in the ventricle.

From what I have said I hope I have made it clear that in a given case of valvular disease of the heart the prognosis depends to a certain degree upon the valve involved and the particular lesion present, but more upon the condition of the myocardium and the coats of the arteries, especially their muscles, and whether they are capable of being developed sufficiently to overcome the valvular defect and carry on the circulation; and that our efforts in treatment should be directed toward instituting measures to bring about these results.

The first of these measures is rest in bed, in order to make the

work of the heart as light as is possible; the second, having the same object in view, is elimination by catharsis, diuresis, and diaphoresis, removing obstruction to the onflow of the blood.

However, rest alone will not restore a weakened muscle to a normal condition; proper food and proper exercise are necessary to complete the cure. When there is permanent valvular defect we must recognize the fact that the defect is permanent and we must bend our energies to devising means for improving the circulation, not merely by improving the strength of the heart-muscle at one point and removing obstruction to the circulation at another, but by improving the strength of the arterial system in general and of the arterioles in particular. Herein lies the value of salt baths, of carbonic acid gas baths, of scientifically administered massage, and of carefully regulated exercises, developing the muscles of these structures and stimulating their rhythmic contraction and relaxation.

By the careful study of our patients and the recognition of the particular lesion presented and the degree to which it has progressed, we are able to outline a plan of treatment that will be of some value and, after observation of our patients for a while, of making a prognosis which will be true.

THE PREVENTION AND TREATMENT OF ACUTE NEPHRITIS

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Notwithstanding the continuous study of diseases of the kidneys by the most skilful investigators with accurate methods during the last quarter of a century, the acute nephritis group still embraces practically the same pathologic conditions that were originally included in it. The older treatment, however, is rapidly giving place to more successful and seemingly more reasonable methods in which greater consideration is had for the inflamed condition of the kidneys, as distinguished from the mere suspension of their function, and to methods that are more in harmony with those applied to other organs when in a similar state of congestion.

A great deal has been added also to our understanding of the causes of acute nephritis, but it seems to me that too little application has been made of this knowledge to the prevention of the disease. Too little attention has been paid, in other words, to avoiding the influences that are recognized as capable of exciting renal irritation during the treatment of other diseases. The very weakness of our efforts to cure the nephritis and the almost uselessness of medication in the worst cases make it all the more our duty to exercise prophylaxis in its fullest sense—sleepless care of the patient in anticipation of danger.

Acute nephritis is rarely, if ever, a primary disease. Sometimes its origin cannot be more satisfactorily explained than by attributing it to cold; but in such instances it is altogether probable that the exposure has acted only as a contributory influence, or, as we generally call it, a predisposing cause, by creating susceptibility that did not previously exist, or by diminishing the power of resistance to the action of some pathogenic microörganism, toxin, or other body or substance highly irritating to the kidneys. Some

writers assume that the cold induces retention of excrementitious matter, others that it changes the character of the albuminous constituents of the blood in such a way as to cause irritation of the kidneys. In some cases, no doubt, the abnormal blood-state results from arrest or pervertion of some process in metabolism with increased production of the purin bodies or other substances concerning which our knowledge is even more incomplete; or it may come from an association or accumulation of these influences.

The most generally conceded, if not definitely determined, cause of acute nephritis is the irritant action of the toxins of the acute infectious diseases in their passage from the body through the kidneys. And of the diseases in which the toxins are believed to induce the renal lesions, scarlet fever heads the list, with cholera and yellow fever less common only as their prevalence is more restricted. In addition to these, there is a long list of diseases, including influenza, the acute exanthemata, diphtheria, erysipelas, infectious jaundice and other diseases involving the integument, sepsis, malignant endocarditis, and in short all the other acute infectious diseases, which are more or less frequently attended with or followed by some form of acute nephritis. In most of the diseases that are characterized by bacteremia—diseases in which the germs get into the blood—the bacteria themselves are believed to be the exciting cause of nephritis, when it occurs, or they assist directly in its production. This is notably true of typhoid fever; possibly it applies also to pneumonia, malaria, cerebrospinal meningitis, syphilis, and most other affections produced by the streptococci and their allies, or by protozoa. The microörganisms act directly on the parenchyma of the kidneys, for acute nephritis is generally a parenchymatous affection, and it is largely through the kidneys that the germs of these diseases are eliminated. To what extent the abnormal states of the blood and the derangement of organs other than the kidneys should be considered as parts of the disease and not merely as incidental to its pathology and symptomatology, it is perhaps too soon for us to decide. The recognition of these facts, however, enables us to adopt measures of prevention to an extent that we cannot do in many other diseases.

PROPHYLAXIS

We protect our acute infectious patients from nephritis by putting them to bed even in the mildest attacks, by confining them in a properly ventilated room of normal temperature (about 68° F., 20° C.), and by keeping them housed until all danger has passed—the length of time depending on the character of the disease, the constitution of the patient, and even more on the condition of the weather. For, if cold is ever capable of producing nephritis or even congestion of the kidneys in a previously healthy person, it must be doubly harmful to one with an elevated temperature and with congested mucous membranes and skin. We further protect the patient by putting him in a flannel gown between woolen blankets in cold weather, and by continuing the warm clothing during convalescence. And of course we must see to it that the reversion from these means of protection is not too abrupt. It is our duty also to explain to the parents and nurses the necessity of these restrictions, for intelligent coöperation is more faithful than blind obedience. In scarlet fever it is important to bathe the body every day and to anoint it with vaseline or oil during the period of desquamation, in order to prevent spread of the infection, but the bathing must be correctly done or it becomes an exceedingly dangerous procedure. We must describe explicitly, therefore, the methods of bathing to be employed. The use of phenolated oil or vaseline is sometimes thoughtlessly permitted, or possibly recommended as being more thoroughly protective than the plain oil. Possibly it is harmless in most cases, but its use is unnecessary and there is a possibility of injurious effect from the absorption of the phenic acid to the extent that it is absorbable by the skin.

Next, we must regulate the diet and secure regular action of the bowels, in order to prevent the formation and accumulation of toxic matter. And, although recent investigation has failed to show that the substances of chemical and bacteriologic formation ordinarily present in the intestines during digestion are toxic to the kidneys, even when they are retained for more than the normal length of time by constipation, it is not safe to conclude that their presence should be disregarded when there are at the same time in the blood the toxins of an acute infectious disease and the products of fever-changed metabolic activities. The kidneys should be relieved of

the unnecessary duty of carrying off the poisons that can be and should be removed through the bowels, whether or not it becomes necessary to supplement their action by purgation.

Milk is the basis of correct feeding during fever, especially in the diseases of childhood, and there is rarely if ever an occasion for either the starvation water-diet or exclusive meat or carbohydrate feeding. Milk furnishes sufficient nutriment in the most harmless form and it is extremely seldom that it cannot be made to agree with the patient. Containing so large a proportion of water, it often affords enough liquid for the needs of the system when it is given in sufficient quantity to meet the requirement for food. As long as the kidneys are intact and perform their function properly, enough water should be given to ensure the removal of effete matter, but enough must not be given to overtax the organs of circulation and elimination. The excessive administration of liquid is harmful to the kidneys and must be guarded against. Unlimited quantities of water are no longer allowed to fever patients; but perhaps we err too often in neglecting to determine the quantity that should be allowed in the individual cases. The quantity is not a definite one, but should be regulated by the circumstances of each case, the presence or absence of edema, the condition of the heart, and the character of the urine as to quantity, color, specific gravity, albumin, chlorides, and other ingredients. In conditions of obtunded sensibilities, as in typhoid fever, there is more danger of giving too little than too much water, for the patient seldom asks for drink, but in many of the other infections, the persistent thirst is liable to cause excessive drinking.

Great harm can unquestionably be done by indiscriminate medication in the infectious diseases. When possible, therefore, we should avoid the use of drugs that are capable of inducing renal irritation; and when their use is unavoidable we should keep a close watch for the first indication of injurious action. This is made the more necessary by the varying susceptibility of different people. I have recently seen melanuria with abundant albumin induced by only five six-grain doses of sodium phenolsulphonate in a septic patient whose urine did not contain albumin before and promptly became free from it after the phenolsulphonate was discontinued. Potassium chlorate is undoubtedly one of the most injurious drugs

that can be administered to an infectious patient, and there is no doubt that formerly it was employed too generally for its supposed benefit to the angina of scarlet fever and diphtheria. Among other drugs to be avoided or to be used with caution are the salts of mercury and lead, phenic and salicylic acids, the oxalates, phosphorus, many of the coal-tar products, arsenic, the mineral acids, turpentine, chloroform, ether, tar, copaiva and other balsams, and a host of secret "remedies," possibly including some of the ready-made foods.

Most cases of acute infection require little or no medication. Those diseases for which there is no specific treatment are best handled expectantly, meeting the symptoms as they occur. In the few for which we have specifics, the renal complications are prevented to a great extent by the early arrest of the infectious process. These rarely occur after the use of antitoxin in diphtheria, and similar claims are being made for the serum treatment of other diseases, particularly of scarlatina. For many years I have made a practice of prescribing an alkaline salt, usually sodium salicylate, in small doses to nearly every child with an acute infectious disease. I have not had a severe case of nephritis in a patient thus treated, although the climatic conditions (in Cincinnati) were generally very favorable to the development of complications. I do not think that I am wrong in attributing part of this good fortune to the action of the alkali, although most of my cases were probably of a mild type. The acidity of the urine is diminished by it; but the essential action of the drug is no doubt farther back, possibly in the increased alkalinity of the blood, possibly in diminished formation of purin bodies in the liver, or merely in a decreased production of the acid phosphates that ordinarily give the urine its acid reaction.

Finally, we should make routine examinations of the urine, not limiting our analysis to the search for albumin and casts, but including the compounds of nitrogen, the sulphates, and particularly the chlorides. A patient should never be dismissed as cured so long as there remains any evidence of renal inefficiency, although weeks may have elapsed since the apparent recovery from the infection. If this course were more generally followed, I believe that fewer cases of unaccountable chronic nephritis would be reported.

The prophylaxis against acute nephritis is by no means limited to the acute infections, although these are by far the most common causes of the acute forms of nephritis. In poisoning by the metals and their salts, for example, and more particularly in lead intoxication, we ought not to throw too much work upon the kidneys by trying to secure too rapid removal of the poison. The iodides should never be given while the sufferer continues exposed or while the alimentary tract contains any of the metal, and they must not be given in too large doses. Greater caution should be exercised, perhaps, in the use of corrosive sublimate, iodoform, and phenic acid, in solution, in substance, or as gauze dressings to wounds and in closed cavities. Of the causes of the nephritis of pregnancy so little is positively known that prophylaxis is limited to regulation of the functions in general. Then there are many injurious practices, such as excessive bicycling and many so-called "endurance tests" that are commonly productive of albuminuria and doubtless favor the more serious lesions of the kidneys. And against all such dangers we should warn the people over whose health we have the supervision.

GENERAL TREATMENT

Our most effective means of combating acute nephritis are warmth, rest, and restriction of diet. If the patient has not been placed in a proper room during a previous illness, with warm garments and bedclothing, these things should be attended to immediately. The temperature of the room should be kept as constantly as possible at 68° F. (20° C.) day and night, but ventilation must not be neglected. The admission of an abundance of fresh air, by preserving the purity of the blood, is one of the most valuable aids to treatment in all conditions, but it is more generally neglected than any other part of the treatment, irrespective of condition and disease. The bedclothing, although warm, should not be too heavy. The greater the edema and the more the urine is suppressed, the greater will be the need of warmth, and as a rule, the action of the skin should be stimulated by additional heat. A hot-water bottle or two applied to the back, over the region of the kidneys, often supplies enough heat to induce and maintain moderate sweating. The patient must not be made too uncomfortable, however, especially if a child, lest he become restless and get chilled by throwing aside

the covering. Hot baths are beneficial only when they can be given without the possibility of exposure. The patient should not be taken into another room, if avoidable, but a portable tub should be employed. Evening is the best time for the hot bath, for the diaphoretic action can be sustained with less difficulty. In adult cases this action can be increased by the administration of a Dover's powder, the resulting sleep removing the danger of exposure through restlessness. When a hot water bath cannot be given with safety, just as good diaphoretic effects can be obtained from hot air, using the bedclothing as a canopy and passing the heated air from a lamp under it through any convenient pipe.

Diaphoresis should be maintained in all cases, and the ease with which it can be kept up is of prognostic significance. The removal of the chlorides from the blood is no doubt of as much importance as that of the substances which we have generally regarded as more highly toxic. In children, the diaphoretic action of the skin can generally be stimulated sufficiently by means of warm clothing and hot bottles or baths, and it is seldom necessary or desirable to give diaphoretic drugs. In adults, however, it occasionally becomes advisable to employ pilocarpin or other remedy of the same class. Pilocarpin must be used cautiously, however, and two or three doses of $^1/_8$ or $^1/_{12}$ grain (0.008 or 0.005 gram), at intervals of a half hour, are safer at the start than a single large dose. The fact that one has never seen its unpleasant effects should not lead him to lay aside this precaution. Sometimes the fluidextract of jaborandi can be employed with greater safety and equal benefit, especially when it is desired to keep up a moderate but uninterrupted diaphoresis. Neither remedy should be employed, of course, in a case attended with marked cardiac weakness or general prostration, and it is usually advisable to protect the heart by a previous dose of brandy, camphor, or strychnin.

The second requisite to treatment is rest, and this should be complete. It should embrace not only confinement to bed and quiet, both of which give rest to the heart and reduce the production of waste, but also the freeing of the kidneys from functional action for a few days, as far as this is possible. We all know that complete cessation of renal action soon becomes harmful; it is one of the results of intense inflammation that we dread most. This fear,

however, has led us into the error of doing too much for its prevention, both in the almost frantic use of drugs a few years ago, and in the giving too freely of liquids. We have now learned, chiefly through von Noorden's investigations, that it is better to refrain from all efforts to force renal action when the disease is severe, and to withhold both food and drink for the first three or four days in all cases in which the starvation can be endured. In case nourishment is necessary to the enfeebled body, and after the preliminary period of abstinence is over, milk alone should be given, and at first only in the smallest quantity that will sustain the strength. It should be given warm, unless it is repulsive to the patient, and pure or in such dilution as the age of a child may require. Previous boiling is unnecessary, of course, and generally objectionable. When pure milk is not readily digested and assimilated, the addition of a little lime-water may remove the objection; this is better, I think, than carbonated water or the so-called "vichy" in all nephritis cases. If cold milk is allowed, it should be given in small quantities at short intervals. Soups, broths, and beef-tea must be excluded on account of the salt and nitrogenous matter contained in them, and as a rule, water should not be given so long as the quantity of milk consumed is less than that required for the full sustenance of the patient. After the renal function has been sufficiently restored, water can be given, and lemonade is often a good and agreeable form in which to give it. It is often more acceptable and better than either plain water or the alkaline solutions recommended from time to time. The chemical composition of the drinking water of a locality is often of some importance in this connection. The old advice to drink freely of pure water is no longer permissible in any stage of acute nephritis. The quantity to be allowed must be modified from time to time to correspond with changes in the renal condition. The "flushing out" process must be deferred until all the more acute symptoms have subsided or until it has become evident that they will not subside. Increased ingestion of liquid should follow, rather than precede, increased excretion, as long as the evidences of renal congestion continue. And later, whenever it is discovered that an increase of the liquid taken is not responded to by an increased secretion of urine, the allowance should be reduced, unless the failure can be accounted for by profuse sweating or the recent action of a brisk cathartic. In the

restriction of drink, however, in cases unattended with dropsy, allowance must always be made for the needs of the secretions other than that of the kidneys, more particularly while cathartics are being administered.

Few drugs are directly of benefit to the inflamed kidneys. Many have been tried and found wanting. A recently reported test of the long discarded turpentine treatment of scarlatinal nephritis in quite a number of cases without great damage demonstrates nothing more clearly perhaps than the enormous odds against which recovery can occur. Improvement continued without interruption, or seemed to be hastened in some cases, and hematuria was induced in only two or three, for large doses were not given. But does not the varying action of this and other drugs suggest another possibility, namely, that we as clinicians are falling behind the progress in pathology? Is it not probable that with greater skill to distinguish the several forms of nephritis we might become able to select remedies that would prove more effective in each form of the disease.

The treatment of every case of acute nephritis should begin with the administration of a cathartic in doses sufficient to free the intestines of possible toxic matter. Free catharsis, indeed, often seems to avert a beginning attack of nephritis. And at any time during the course of the disease, if the suppression of urine becomes great, and when edema appears, a brisk cathartic such as magnesium sulphate in concentrated solution should be given. Even when there is little or no edema, this will sometimes reduce the congestion of the kidneys to such an extent that their function is reëstablished and the tubules, if obstructed, are washed out in the most natural manner. Following this, particularly if there is thirst, a little more allowance of water may be granted, or a mildly diuretic draught may be given if the stimulation of renal action seems important. But it is better, as a rule, to continue our efforts to remove the toxic products from the blood by means of cathartics and diaphoretics as long as the acute symptoms persist. Any of the laxative salts of sodium or potassium are safer than the mineral waters containing sodium chloride or the various ready-to-use preparations on the market, abounding in "antacid, alterative, cholagogue, deobstruent, diuretic, *secernent*, laxative and cathartic properties." Calomel should probably be used with caution in acute cases; its diuretic action is most available in diseases of the heart.

The various symptoms are treated for the most part as they would be in other circumstances, with the restriction of remedies to those that are least harmful to the kidneys. Fever seldom requires special consideration except as a part of an acute infection. Aconite possesses peculiar advantages in some instances, especially when the arterial tension is high, but it must be used with great caution. Glonoin in minute doses at short intervals is better for the reduction of arterial tension; some prefer the nitrites, the liquor ammonii acetatis, etc. But all these drugs are more useful in cases that are becoming chronic. For restlessness or headache, and to avoid the use of opium, strontium or sodium bromide may be given by the mouth, or chloral by the rectum. Vomiting is sometimes troublesome and calls in turn for granulated ice, hot water, brandy, sinapisms over the stomach, etc. A little vinegar or a mineral acid sometimes arrests it, but in many cases a profuse sweat induced by a hot bath or hot pack is the best means of relief.

Counterirritation by means of a dozen or more dry cups applied to the region of the kidneys and removed before stagnation of the blood can occur is sometimes followed by improvement of the renal condition. Other counterirritants may be employed, but of course not such as turpentine or cantharidis or other agents that are likely to increase renal congestion if absorbed into the blood.

Venesection is periodically advised, especially in cases of approaching uremia or cerebral edema, but it is probably too seldom performed. After the bleeding, the loss to the circulation in blood withdrawn should be restored by the introduction of water through intravenous injection or hypodermoclysis with decinormal salt solution or by means of prolonged rectal irrigation through the long tube with warm sterile water.

When and to what extent we should employ diuretics in the treatment of acute nephritis is a question for serious consideration in a resisting case and one that is often difficult of answer. There can be little doubt that diuretics were formerly employed too freely and perhaps with too little discrimination. In the early treatment of a case there seems to be no special indication for their use, and in a severe state of inflammation they may prove harmful. But success in treatment does not come from too close adherence to fast rules. While the heart is normal in force and action, water remains the most serviceable diuretic, but when the heart's action is deficient

and the blood-tension is low, digitalis is generally indicated, providing that the heart weakness is not due to advanced myocarditis. The fresh infusion of the English leaves is generally the best preparation; but sometimes it cannot be retained by the already disturbed stomach and then the powder or tincture must be employed, preferably in capsules with as little water as possible. The selection of the remedy should depend, however, on existing indications. Strophanthus does not always prove effective when the arterial tension is high, and sometimes a combined preparation seems better suited to the case. Great harm may be done, however, by the indiscriminate addition of strychnin to "heart tonic" prescriptions for nephritic cases. The diuretic action of these remedies may be increased by the simultaneous or subsequent administration of theobromin-sodium salicylate. More energetic diuretics are seldom useful in acute nephritis, and during convalescence especially there is often nothing better than the potassium tartrate solution (a drachm in a half pint or more of hot water, with a little lemon juice added, drunk leisurely after it has cooled).

In convalescence, the restrictions of food should not be withdrawn too rapidly, and the effect of each addition to the diet-list should be carefully watched. Unsalted food is extremely unsavory, but salt and spices must be restored with caution. My experience with the salt-free diet has been limited, but exceedingly satisfactory, more especially in chronic cases, however. As improvement becomes more firmly established, easily digestible carbohydrates may be allowed, but meats should generally be withheld until the last. Most persons can eat eggs with impunity, short of excess in quantity and wrong cooking. It is at this time generally that iron is most needed, but the need of it should be determined by examination of the blood. The preparation employed is probably a matter of personal choice. The tincture, especially when given in milk, has seemed to be more uniformly acceptable and beneficial in my cases than the liquor ferri et ammonii acetatis.

A patient should be given the benefits of sunlight. Until it is safe to permit him to go out of doors he can sit or lie near a window facing southward. It is advisable, indeed, to have such exposure during the entire illness; and, for patients who can avail themselves of such advantages, when recovery is persistently deferred, nothing is so good as a prolonged visit to a warm, equable climate like that of southern California.

THE TREATMENT OF MIGRAINE WITH SPECIAL REFERENCE TO THE USE OF CANNABIS INDICA

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Of Paris

An attack of migraine is simply a clinical complexus, a manifestation of a pathological constitutional condition. During the intervals between attacks the subject is a patient, whatever may be his apparent state of good health; and it is during his intercalary periods that he ought to be treated in order that his original predisposition may be modified and that secondary causes that give rise to the attacks may be averted.

The treatment of such a patient should comprise three parts: (1) That of the constitutional, neuro-arthritic condition; (2) the organic or functional disturbance, to be found in every case; (3) the causes that give rise to the attack.

I

Each patient who suffers from migraine is an arthritic person in whom the diathesis affects more specially the nervous system. In order to modify this general constitutional condition and its special localization, the association of an appropriate diet, with the systematic use of cannabis indica and of hot douches, often gives good results.

(A) The usual antiarthritic diet has a twofold purpose: to lessen uricemia, and to prevent auto-intoxication of digestive origin.

It is indispensable that such patients should drink nothing but pure water or hot infusions; alcohol is forbidden, in whatever form, and it is quite singular to note how sensitive to the smallest amount of alcohol these persons predisposed to migraine can be. Nothing but absolute moderation will do; all the great sufferers from migraine who have been cured had to become water-drinkers. With the midday meal tea is an excellent drink, digestive, stimulating and diuretic; Ceylon tea, weak and very hot, is to be pre-

ferred, made at table with boiling water and without being allowed to stand. At dinner, pure water is best, or some diuretic, slightly mineralized table water, such as Alet, Thonon, Evian.

Meats allowed are beef, mutton, or poultry, always well cooked, and grown animals in preference to young. All sorts of vegetables can be permitted, starchy or green, with the exception of those that are notoriously acid; they should be taken well-cooked and best done in the form of purées; as dessert, fresh cheese, milk puddings, ripe fruit, either fresh or stewed.

In addition to this habitual diet, when the migraine patient is in a period of successive attacks he should be put on a still more restricted food-programme: no meat at all; but milk and vegetables, eggs, and farinaceous foods. Thus, for early breakfast, half a liter of milk with a little coffee; at noon and in the evening, a milk soup with tapioca or semolino, mashed potatoes or lentils, macaroni or chiccory, a milk-and-egg pudding, and milk to drink. If the patient feels hungry in the afternoon, he can take a cup of chocolate prepared with milk.

(B) It was Dr. Stephen Mackenzie, of the London Hospital, who called attention to the great usefulness of cannabis indica in the treatment of headache. "The effect of this drug," he wrote, "as an analgesic is specially noticeable when we have to treat pain combined with spasmodic symptoms. Its action is favorable in all forms of cephalalgia, whether purely functional in nature or due to an organic complaint,—even in cases where the pain is due to cerebral tumor or chronic uremia. It can stop a migraine either at the start, or after it has lasted a certain length of time; its prolonged use is capable of lessening the frequency and intensity of attacks of migraine.

"There is a special form of cephalalgia to which cannabis indica acts as a real specific. This cephalalgia is characterized by its continuous or sub-continuous type. It commences on awakening, and lasts the entire day; it may disappear toward evening, but if the patient gets up during the night, it occurs afresh. It is generally a diffused pain of the entire head, ill-defined, increasing at certain moments, and capable of lasting for weeks, months and even years. Although not violent enough to interfere with occupations not requiring great intellectual efforts, it is still a source of constant

discomfort, and puts the patient to serious inconvenience. This form of headache can be observed in either sex, the patient being generally young or middle-aged. Its causes are obscure. Many persons who suffer from it show no trace of anemia; again, when this morbid state does exist, its treatment has no influence on the headache. Nor have I been able to detect any connection between this form of headache and the presence of an excess of uric acid in the blood.

"This variety of cephalalgia is almost always curable by cannabis indica, either given alone or combined with other remedies suitable to counteract other concomitant troubles. I generally give cannabis indica in the form of the extract, first ¼ grain and then ½ grain, in a pill, morning and evening. If improvement manifests itself, I continue at the same doses, but when at the end of one or two weeks there is no change in the patient's condition, I increase the dose and give a grain in the evening and half a grain in the morning. If this dose is still insufficient, I give a grain night and morning. In particularly stubborn cases I raise the dose steadily (always giving the larger dose in the evening) until I obtain either marked relief, or symptoms of intoxication appear."

Mackenzie reported only a few cases of slight intolerance. When this paper came to my notice I at once put the treatment to a test in the case of a patient who for several years had suffered from habitual morning headache, with frequent acute attacks,—a desperately stubborn case that had resisted a variety of hygienic and pharmaceutical combinations. The patient was a physician who for the two preceding winters had suffered from such continuous headaches occasioned by his regular professional work,—night visits, for instance,—that he was beginning to wonder whether he should be able to continue in active practice. During the whole month of October he took at bed-time every evening a ¼ grain pill of the hydro-alcoholic extract of cannabis indica, and during November, when his practice began to increase as the season set in, he had the satisfaction of finding that his daily morning headache was less and his acute attacks not so frequent.

For eighteen consecutive months he took systematically every evening one of these pills, doubling the dose during such times as he could not avoid being exposed to the usual causes that brought on a

migraine. The drug was always well borne and gave rise to no symptoms.

It is necessary to have oneself felt the torment of this daily morning headache on awakening and of this constant threatening of an atrocious attack of migraine with its vomiting and other symptoms, to realise the intense relief experienced by this physician when he felt finally certain that he was not a victim to a mere lull in the disorder, as had happened on former occasions. For this reason he has now continued to take the cannabis indica for a number of years during the periods when he is most exposed to the causes that had previously been productive of an attack.

Migraine cases are common in practice. During the last ten years I have had many opportunities of prescribing this treatment, and the favorable results given by the cannabis indica have seemed to me sufficiently constant almost to warrant the epithet of a specific remedy given to it by Mackenzie.

I use quarter-grain pills of hydro-alcoholic extract of cannabis indica. The extract of cannabis indica of the French Codex is an alcoholic one obtained by treating powdered cannabis with 60° alcohol, then distilling the alcohol and concentrating the remainder to a soft mass. After a certain length of time the extract separates into two parts, a green resinous portion at the top, and a brownish portion, very soluble in water, at the bottom. Since the active principle is contained in the resinous portion, it is easy to realise the wide variations reported in the action of hemp extract, according to whether an extract recently prepared, consequently homogeneous, was employed, or one that had been kept long enough for the separation to take place, when the upper portion would be very active and the lower one little or not at all active.

One pill should be given at bed-time every evening for thirty consecutive days; at the end of this time the improvement is usually noticeable, and the same dose can be continued. If one pill proves insufficient, double the dose for two weeks, but give only one pill for the following two weeks, alternating in this way from two weeks to two weeks. If in spite of this the headache still resists, give two pills at bed-time and one in the morning.

These low doses are efficacious if the extract is of good quality. It is not large doses that succeed, but the steady and prolonged use

of the drug given systematically for an entire year, even though the patient appears to be in the best of health and the attacks are less frequent. During the years that follow, the patient can take treatments of one month, separated by more or less prolonged intervals. It would seem that we must modify a constant and special state of hyperexcitability of the nervous system by some permanent means of control, and that in cannabis indica we have such a means, and one as efficacious as it is harmless. It is probable that a migraine patient should never abandon its use completely, but return to it deliberately as a preventive at such periods,—and particularly in winter,—and under such circumstances as his experience has shown him to be particularly exposed to his painful attacks. At the doses in which I give it I have never found a patient who could not take it, and I know of no contra-indications to its use; it has no effect on the digestion, does not constipate, and rather stimulates appetite. Again, the system does not appear to grow accustomed to it, so that the drug loses its effect, as is the case with all sedative remedies of the nervous system. I have migraine patients who after having taken hemp for several consecutive years without its having had any undesirable action on them whatever, still reacted perfectly well to the dose that succeeded with them at the outset, whenever they institute a short preventive treatment anew.

(C) The warm douche. The douche suitable for neuro-arthritic patients with migraine should be at the same time tonic, revulsive and sedative; the cold douche is too stimulating; it is better to give the hot douche alone or to combine it with a cold one.

The hot water should be used first, at 38 to 40° C., rising to 43 to 45° in one or two minutes, in a broken stream, touching neither head nor neck, and ending in a full stream on the feet. As soon as the patient becomes used to it the final temperature of the water should be lowered, until the douche ends in a brisk application of cold water. With patients thoroughly broken in to the treatment the temperature of the water can be gradually raised from 38° to 45 to 46°, and then suddenly a strong stream of cold water can be dashed on the entire surface of the body, ending in a full cold jet to the feet.

This should be done every day, no matter at what time of year, without any interruption, for consecutive periods of one or

two months. It is very important to exact the daily douche. The first ten usually produce a feeling of fatigue; it is only when this has been gotten over that any benefit is experienced, thirty treatments on thirty consecutive days are always useful, but the same number spread over sixty days do not succeed, and less still, of course, if only taken two or three times a week. In the latter case they cause only fatigue, discomfort, and disappointment, without reaching the point at which the patient gets into training, so to speak, a point indispensable in hydrotherapeutics.

Each douche should be followed by a vigorous rubbing—first with a flannel mitten and finally with a hair mitten, as soon as the patient can stand it.

As is the case with the hemp, the douche treatment is not a casual but a fundamental remedy, and must be included in the general plan for the curing of the disorder. Several series must be taken each year. Like the cannabis indica it has the precious advantage of being always efficacious and of not losing its favorable effect, even in the case of patients who have had recourse to it for several years in succession.

II

Every migraine patient, in addition to his arthritic constitution, suffers from some functional or organic disorder of some organ, varying according to the individual: stomach, liver, kidneys, etc., and it is fully as important that this disorder should be treated as it is to look after his general state. It is not easy, *a priori*, to appreciate the connection between these sometimes trifling functional disturbances and the patient's severe periodical headaches. Yet experience proves that this connection is a real one, and that the most logical combinations of a general order are insufficient if we do not ameliorate this local disturbance which one may discover in each migraine patient. One should first investigate the digestive tract, stomach, intestines and liver; for without accepting the opinion so generally credited, that migraines are in all cases a manifestation of dyspepsia, one must always bear in mind the frequent correlation of migraine with disordered digestion, particularly in cases of gastric hyperesthesia, mucomembranous enteritis, habitual constipation, and all hepatic disorders. But many are the instances of migraine which resist all anti-dyspeptic treatment.

With women, and even with young girls, after the digestive tract come the uterus and its appendages, and this connection of the organs of reproduction with migraine is of real practical importance. How many women are there not who, quite well during the intermenstrual periods, cannot have their menses without more or less headache, sometimes slight, sometimes regular migraine. So true is this that a headache at such times is so common that they think it the necessary accompaniment to the monthly flow, do not complain of it, and make no effort to put a stop to a symptom which they look on as inevitable. One may in such cases be quite sure that he will find the cause of the headache in the uterus or ovaries. The lesion will almost always be an unimportant one, to which the patient has never called attention and which has to be looked for to be discovered: cervical stenosis, endometritis, endocervicitis, prolapsus, subinvolution, dysmenorrhea or vulvo-vaginitis. The criterium is supplied by the result: rapid modification of the headaches following local treatment.

The most singular disorders may be the point of departure of a migraine; thus a case is cited in which dilatation of a urethral stricture cured a man who had suffered greatly from migraine. Later on the stricture reformed, and the headaches commenced again, to be once more relieved by urethrotomy.

I once took care of a young patient of sixteen, who suffered severely from migraine, and in whom I found partial nasal obstruction, a spur from the septum obstructing the left fossa, while the lower right turbinal was hypertrophied. When the spur had been removed, the membrane cauterized and free breathing restored, the headaches disappeared.

In other cases the source of the headache will be found in the eyes, errors of refraction, astigmatism, overwork, myopia, etc. The possibility of this source of the headache should always be borne in mind, and the eyes most carefully investigated.

III

Persons predisposed to migraine should make every effort to protect themselves from the causes that give rise to the attacks; they must study themselves closely to detect these causes, as they are often very trifling and differ in each person.

Some are connected with the digestion,—imprudences, constipation, etc.; others depend on the weather, cold, or wind. Migraine patients often suffer most in winter, particularly when the weather is dry and invigorating; their heads are very sensitive to cold, even asleep in bed. On awakening, and before the curtains are drawn, they know that snow has fallen, on account of the feeling in their head. One patient who goes to bed well in a warm room, will wake up with a headache if the room has grown cold. On several occasions I have succeeded in relieving such patients from their matutinal headache by advising them to wear some head-covering all night.

Others complain of the influence of cold wind, particularly in high altitudes. I have seen patients who could not stand a mountain climate at all; as soon as they felt the fresh cold air coming off the glaciers they were seized with painful sensations in the skin of the forehead, followed in due course by a full attack of migraine. The same susceptibility manifests itself in some physicians who cannot go out at night in winter without paying for it the next day with a certain degree of headache.

With other patients the morning headache is the expression of an intoxication to which they have been exposed during sleep; lack of fresh air in their bedroom, or reflux down the chimney of gases coming from neighboring chimneys through fissures. The vitiated atmosphere of theaters and ballrooms, and tobacco smoke in fumoirs, have the same effect. Some persons predisposed to migraine can sit up late working at home without any ill effects the next day, but if they spend the evening at the theatre or dining with friends, they return in a state of intoxication through smoke and bad air, and have an attack the following day.

Tobacco is in many cases a poison quite as active as alcohol, and is always advantageously suppressed.

The normal menstrual congestion, prolonged mental preoccupations, and anxiety or trouble, are all pretexts that suffice to bring on an attack.

IV

Treatment of an Attack.—After a night of normal sleep a patient wakes up ill-disposed, his usual morning headache is more marked, the general heaviness in the head is soon accompanied by

a sensation of cold, vertigo, disordered vision, and bad taste in the mouth, etc.: such are the usual signs that forecast an approaching attack. Can we at this stage hope to ward it off? I have practically abandoned all forms of treatment by the mouth, which have only given me uncertain results and seemed to upset the stomach and hasten the appearance of nausea. But I have often observed good results from a hot douche taken at once, as soon as possible after awakening, by a person accustomed to that treatment. The douche should be given in the way described above, and should be essentially sedative, very hot and prolonged (two minutes), with little pressure, and applied particularly to the lower half of the body. This often succeeds in breaking up the attack, and the patient then is aware of it at once. He feels a sensation of relief; calm and nervous relaxation come over him, whereas previously he had been as it were poisoned; his oppression departs and fresh physical and intellectual vigor manifests itself. If, in addition, he is able to rest for the remainder of the morning, avoid the influence of cold, moderate his professional work and thus await the hour for lunch, he will have a fair chance of escaping the full attack. Often, after having eaten, he will be agreeably surprised to find that his head is quite free from pain.

But if the attack proceeds and reaches its full development, there is no use opposing it. All that can be done is to moderate the sometimes atrocious suffering; rest in bed in darkness, and applications to the forehead of the following preparation:

Menthol		
Chloroform		
Salicylate of methyl, of each	5.00	75 grains
Lanoline	30.00	1 ounce

Applications of this can be alternated with very hot dry, crash towels, wrapped around the entire head and renewed as soon as they are no longer hot, every five or ten minutes. Finally, during the paroxysm of pain, especially toward night, the following enema will secure rest:

Chlorhydro-sulphate of quinine	0.50	8 grains
Antipyrin	1.	15 grains
Laudanum	0.60	10 minims
Boiled water	30.	1 ounce

During this artificial sleep the migraine goes through its evolution, and the patient finally awakes very much relieved or even cured.

V

Treatment by Mineral Springs.—The treatment of migraine as above outlined should be complete during the summer season at certain mineral-water resorts. Migraine patients require each year a period during which they give up entirely their professional or social occupations; and they derive advantage from doing this in places where at the same time they can profit by a mineral water-cure suited to their condition, a treatment always useful, and in some instances more efficacious, in spite of its brevity, than prolonged courses of treatment at home. A resort should then be selected among those whose general effect is anti-arthritic, and in particular one whose functional specialty is that of disorders of the organ habitually involved in the case in question.

Vichy, where the treatment is above all else anti-arthritic and anti-uricemic, is frequently indicated. Patients adapted to it are those with dyspeptic migraine, of gastric or hepatic origin; they will find in it, for their diathetic condition, the most active modifying elements, and for their digestive disorder, a most excellent treatment whose efficacy no longer needs to be proved.

A uricemic patient, with whom there is as yet no marked localization, but who is shown to be such by the urates eliminated in the urine, will also benefit by a season at Vichy, and during the winter that follows it will prove that his headaches are less severe.

With children of gouty parents, where the heredity appears in the form of headaches, and also with neurasthenic patients whose system is below par, Royat is more specially indicated.

Patients with gout or gravel are best sent to such resorts as Contrexeville or Vittel; if at the same time they need sedation of the nervous system they will find at Evian or Thonon, together with a method of blood-purification, a particularly agreeable abode in very pure air, in a climate that is essentially sedative and tonic.

With uterine forms of migraine Luxeuil or Saint Sauveur are always beneficial; by means of their action in lessening conges-

tion of all the organs of reproduction, and by their general sedative effect and toning of the nervous system, these two resorts procure for these patients, who suffer possibly more than we believe, permanent relief and help to increase their strength of resistance.

When the migraine patient is an overwrought brain-worker, a victim to a liberal profession, or a neurasthenic "by excess," he should go to Saint-Gervais or to Bagnères-de-Bigorre, where he will find, at a moderate altitude, an atmosphere that is stimulating without excitement, a water slightly laxative, and sedative baths, together with other elements calculated to reëstablish the balance in disordered nervous systems.

THE PROPHYLAXIS OF NERVOUS DISEASE WITH SPECIAL REFERENCE TO EDUCATIONAL INFLUENCES IN THE GROWING CHILD

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The prevention of nervous disease consists in the prevention of other disease and the building up of vigorous health. The trend of modern life is partly toward, partly away from, such prophylaxis. The infectious diseases play no small part in the production of both organic and functional nervous disease. Sanitation has achieved much in the way of removing or lessening these infections, and thus in lessening the cause of nervous disease.

But influenza, productive of so many nervous disorders, because so widely prevalent, yet evades the sanitarian, and many of the common infections still play havoc with life and nerves. Syphilis, the cause of so many organic nervous diseases, alcohol, and excesses of all kinds, are etiologic factors of great moment, and play their part in the present as in the past. Diseases of blood-vessels, Bright's disease, etc., which so often lead to brain disease, are, perhaps, on the increase. The cause of this, as of the increase of nerve disease itself, is largely in the mode of modern life.

The rapidly-multiplying factories, the marvelous growth of cities, the high tension of modern life, all tend to increase disease, especially nervous disease, a tendency that is only strengthened by the great care given to the weakling and his multiplication among men. Against all this the physician can only contend by preaching saner modes of life, above all a simpler life with simpler needs.

Among the most common causes of nervous disease are the accidents of life, trauma, with its devastating effects upon mind and nerves, and all that arouses the depressing and painful emotions,—for cankering care, grief, terror, anger, disappointment, humiliation, and the like, unfortunately only too often produce serious

nervous disorders. Even here the tact and experience of the physician may do much to ward off threatened disease in the individual case.

But the one fact that confronts us in relation to all the aforementioned etiologic factors is that, whatever the exciting cause, nervous disease is most likely to occur in those predisposed to it. This predisposition may be the result of intrauterine disease, or of disease or injury in the early years of life; but mostly it is an hereditary taint. It is, therefore, the duty of the physician to do what he can to lessen the marriage of the unfit. But we cannot hope to achieve much in this direction, and the problem remains with us, what to do with the multitude of beings coming into the world whose heritage is nerve weakness.

I believe in the whole field of medicine there is no greater appeal to the services of the physician, nowhere a higher call of duty, than in helping to ward off disease in these little ones and assisting them to grow into healthy men and women.

We may say, then, that prophylaxis of nervous disease consists essentially in the proper training of the child. For this reason it is paramount that the physician devote much thought to the subject of training, especially that of the child predisposed to nervous disease. The subject of training is as large as human nature, and on this occasion I can only touch upon such aspects of it as confront us prominently in the history of the predisposed. Let me approach the subject by taking up a common instance, an hysterical woman and her child. The mother constantly parades her symptoms before the child. Now the instinct imitation, which in education and training is the most powerful influence in the child, and, to a lesser degree is influential throughout life, is prepared to arouse similar manifestations in the child. Furthermore, such a child is ofttimes delicate, likely to present some ailment or complaint, and the latter is just as likely to meet with expressions of sympathy or alarm which only fan the flames. There is already the tendency to self-consciousness and hypersensitiveness in the child, which the manifestations of sympathy unduly exaggerate, preparing it for the outbreak of nervous symptoms. Such a child—this is, of course, true of other children, also,—should be taught to bear trifling ailments patiently and heroically.

A little one in the environment we are depicting, is likely to have a further handicap in the mode of discipline to which it is subjected, sometimes neglect, injustice, or cruel harshness; sometimes over-indulgence, flattery and the like; and, again,—and this is the worst of all,—vacillations between these extremes. No worse environment could be found for the child, no surer foundation for future nervous disease.

When these conditions cannot be remedied the physician should use his influence to remove the child from the parental roof. But a presentation of the facts to the parents may lead to a betterment. The mother may succeed in controlling her manifestations of disease, which would be a great benefit to herself as well, and may undertake the proper training of her offspring.

Now what is the proper training of such a child? We must remember the common tendency to egotism, undue self-consciousness, hypersensitiveness, and oft-resulting morbid introspection; and one of our main objects must be to combat it. No hot-house culture, balls, theaters, etc., which lead to sentimentality and undue sensibility. Flattery, the foolish extolling and exhibiting of the child's supposed shining qualities, overindulgences, all that tends to make the child vain, self-conscious, and selfish, are very harmful. The spoilt child not only loses in character, but becomes an easy prey to "nerves." Neglect and harshness are also prone to cause hypersensitiveness, and lead to morbid brooding.

It is fortunate that strong emotions are given an external discharge in tears, in words, in actions, or what may be. Their undue harboring produces an unpleasant mental state that may lead to unfortunate traits of character, or weaken the nervous system.[1] Emotional conditions which are cherished in secret as often result from unjust treatment; they often end in morbid brooding, and may prove disastrous to the nervous system.

[1] Great outbreaks of passion occur in some children, which may not be without serious consequences. I have succeeded in removing this condition in a number of instances by trifling measures, partly suggestive, partly disciplinary. In a case of a paroxysm the parent was instructed to be quiet and calm, instead of greatly excited as hitherto, and to press upon the child a dose of some badly tasting drug, as milk of asafœtida, assuring the child the latter would strengthen his nerves, so that he could conquer himself, and not have attacks in the future.

The question of punishment of children is often a serious one. There may be in it the element of injustice (or it may appear so to the child), with its damaging effects, and in addition the element of fear. Fear is by all means the one thing that plays most havoc with the nervous system of the child. Certainly punishment has often proved disastrous to nervous children. There can be no doubt, I believe, that punishment is always unfortunate when given in anger, when it is likely, too, to be the expression of anger. To the child it should never mean but one thing,—necessary discipline. Upon some nervous children it were well that any punishment should be inflicted with greatest caution; but it may be said of all children that they should see behind the punishment the parent's love. Love counts more than all else in training a child to character and health and life.

The best means of combatting directly the tendency to hypersensitiveness and introspection is to keep the child busy, to keep its hands and thoughts employed, and away from self; above all to build up a strong unselfish nature.

In addition to what I have already mentioned, we often find in these children, as, also, in others, a tendency to make too much of the trifling ills of life, impatience perhaps, lack of cheerfulness, and the beginning of that worrysome disposition which is a source of constant unhappiness, as well as predisposes to nervous disease. Let us consider how to combat and conquer these tendencies, which may mean so much to the child.

I have already spoken of teaching the child to bear trifling ailments uncomplainingly, rather than encouraging him to make much of them. There are, also, many things which are disagreeable to the senses, or painful, or a mental or physical trial, which the child should also be taught to bear, and bear uncomplainingly. Such a habit might be built up by special effort, by gymnastic training in that direction, if I may so speak. I mean special pains should be taken to teach the child to bear hardships, to be courageous to have a rugged, hardy nature, by leading him to practice doing things that are unpleasant to him, or painful, or difficult, or courageous. A child might in the beginning be led to such acts, which he would instinctively shirk, by mere encouragement, or telling him their purpose, or through the emulation of the acts of his

companions, or by firing him with the story of one of his heroes, or from the love, or for the benefit of, one of his loved ones. And the more frequently he has done such an act, be it to bear pain, to do something he dislikes doing, to conquer his timidity in doing a bold deed,—in other words, the more strongly he has built up a habit of hardihood and courage, the more easily such things come to him, and the better for his future life and health.[2]

In the regulation of the activity of the nervous system no function counts for more than inhibition; and in life and character no trait counts for more than self-control, none is of more value in preventing nervous disease in the predisposed. All that I have said of the cultivation of other traits has been said as well of the cultivation of self-control. But we cannot begin with the latter at too early an age. The modern method of training a child from the moment of birth, not heeding its every cry, appears to be a lesson in self-control, and not unlikely counts in the future training. The earliest conscious lesson of this kind is teaching the child obedience, an extremely valuable lesson to him. At the same time obedience should never mean complete subservience to the will of others, which only weakens character.

In speaking of prophylaxis I cannot omit mentioning such great matters in health as pure air, out-door life, nourishing food, abundance of sleep, and freedom of the play impulse, but their value is so patent that I need not linger here, and will pass on to a subject which plays so large a part in the health of the child,—the school.

The school should be a benefit to the child physically as well as mentally, so that he comes out of it with increased health and vigor. But the more common experience is rather the contrary,—that is, that it mars his health. This is especially true of the neuropathic child, the one with an hereditary taint. He needs the training as much as the vigorous child, and, if it be given in a way befitting him, he should only be benefited by it. But this training should be

[2] In regard to such discipline, it is well to bear in mind that the handicap in life, so far as life spells health and character, is often with the apparently favored classes, those of wealth and rank. In the autobiography, just appearing, of Carl Schurz, that hero of many a hard-fought campaign on battlefield and political arena, says: "I consider myself fortunate in having grown up in simple and modest circumstances." The lives of countless men who have attained to greatness, as well as our daily experience, tell us a like story.

given under lower pressure, if I may so speak, with less tax upon both mind and body than the strong child easily bears.

Let us consider some of the ills to which the school child may be subjected: First, there is the ventilation. Too, often,—probably this is the rule,—the air is very foul. Even under the most favorable circumstances the air is likely not to be as good as it should be, so difficult is proper ventilation. For this reason, if for no other, long hours in the schoolroom are unfortunate.

School hours, too, often lead to grave faults in diet: a breakfast hastily swallowed, or none at all; cold or unsuitable food for lunch, often miserable "truck" eaten between meals; and the same hasty hours may lead to the child's neglecting his bowels and becoming constipated. All these are strong factors in causing physical ailment, poor nutrition, and subsequent nervous disease. One should bear in mind, too, the bad habits children may acquire from one another, seriously endangering their future health.

The undetected and uncorrected defects in the eye, astigmatism, etc., help to the development of nervous symptoms, while unsuitable desks and benches too often lead to spinal curvature.

These and many other factors in school life, which I will not take the time to mention, often play havoc with the child's health and come into prominence in studying the prophylaxis of nervous disease. But I wish to dwell, rather, upon some faulty methods in the school, which seriously threaten the predisposed child.

Overwork, though in itself not usually so harmful to adults, may do serious damage to the impressionable organism of the growing child. There is a prevailing opinion that our schools burden the child's mind more than is good for it. We should be especially careful not to tax the mind of the neuropathic child excessively.[3]

Almost as important as the amount of work is the kind of work done. Pestalozzi, the father of modern education, taught that edu-

[3] Many of our best educators believe that the schools have too long and continuous hours of work, and mistakes are often made besides in giving the child work at home in addition. Shaw says a child should never take work home before his seventh school year. The child is often burdened, too, by the parent's giving it music and extra studies at home. Great harm is often done in this manner. On the contrary, some housework at home often benefits the child. The principal of a Massachusetts school said that those who do some housework at home are more thorough in school. Such assistance at home often helps to health and character.

cation consists in developing what is in the child, not in crowding his mind with a mass of information. This truth seems often to have been lost sight of since. Many years later Herbert Spencer said that the child was injured by being thwarted in its natural inclinations, coercing his attention to books, and giving him subjects before they are understood.

I believe the best educators of the day are trying to come more and more to Pestalozzi's ideas of giving only subjects which accord with the child's inclinations and development. Young children learn languages easily, but they should be taught by speaking them, not by teaching the science of language. Pestalozzi did not even teach the young children to read, but rather to observe carefully and then to describe accurately what they saw, thus exercising them in language and the power of observation.

And so young children should be taught concrete number relations, and not the rules of arithmetic; to draw freely with the pencil and not to write with the pen. All this is to teach the child what he can fully understand and what accords with his inclinations. To-day it is becoming almost universally recognized that nature study and manual training are especially suitable subjects for young children. Such abstract studies as grammar should rarely be taught before the child is 14 years old.

A prominent educator says that that work leads to the wholesome development of the child which maintains his interest in his work, fosters his spirit of inquiry, and satisfies his love of activity. The true test of the work is that it gives joy to the child's heart. The suitable choice of work is especially important to the neuropathic child. For subjects which do not accord with his mental development, and which he does not understand, are a special strain upon his nervous energies, and may become disastrous.

It is not alone the school work that counts with the child's health, but ofttimes even more the emotional conditions which arise in the schoolroom. Many of these are exceedingly harmful to the child. Prominent among causes of such conditions are the examinations to which school children are subjected. It has been observed that a large proportion of children lose weight during examinations. The worry and strain produced often do untold mischief. To a large extent such examinations can and should be abolished.

Then, again, children are often unduly stimulated by bestowal of class honors, prizes, etc. The excessive work entailed often leads to exhaustion, besides which the prize may weaken character by increasing pride and vanity. Such emulation does harm to many in an opposite manner, by the disappointment of failure, and the deep mortification of conscious inferiority.

Another frequent source of harm to nervous children is the punishment inflicted upon them. The mortification and humiliation thereof, as well as the fright and fear, may play havoc with the child's nerves. In this instance the influence is not only in the discipline, but, also, in the teacher who applies it.

Pestalozzi lived with his school-children. They came to love, almost to worship him. He said he could punish without doing harm, in the way of producing bitter feelings or otherwise, for his pupils knew it was the hand of love that was laid upon them.

The teacher means so much to the child, not only in the effect of the discipline upon him, but also in her understanding all his needs and possibilities. There is very much, indeed, for the child's mind and health in selecting the right teacher. But even then, there is so much in the teacher's duties that are a strain upon and overtax her, that she is not always in condition to exert the best influence upon the child.

It will be a great blessing for teacher and pupil alike when the importance of the health and education of the children shall so impress itself upon the public mind that abundant means shall be forthcoming, and good and sufficient schools, and enough teachers, so that the latter will not be overburdened, nor the school-rooms overcrowded. It will, furthermore, be a blessing to the neuropathic, in whose behalf I am now writing, if by methods of grading or classification, those children who would be benefited thereby are put upon shorter hours, less straining work,—upon a lower-pressure system, as I expressed it,—than the average child.

It may be well to speak briefly of some other educational influences within and without the school. The cultivation of the esthetic part of our nature is a very important part of education. The appreciation of all that is beautiful in the world adds so much to life. At the same time, it is well to bear in mind that too much devotion to music has wrought damage to many a weak, nervous

system; and that the ofttimes forced study of one with no inclination to it, is at least an injustice to the child, if it does no further harm.

The literature a child reads is of great significance to its mind and health. That which gives food to its imagination and arouses high ideals is a blessing. But unfortunately not all is of this character. Much of it is weak, some is unseemly, altogether unfit for a child, and some inspires fear to a hazardous degree. I suspect it is not generally appreciated how much fear harms the nervous, both young and old. From the story of the foolish nurse girl to appall her charge, the fear inspired by punishment in home and school, and too often by the stories of religion, horror of the stories in the sensational newspaper, and from the unavoidable accidents of life, there is everywhere abundant opportunity for its blighting effects.

There is another factor in reading that also counts for much; that is, suggestion. Now that our attention has been called to the effects of suggestion in medicine, our daily experience but increases our knowledge of its power. It counts in training[4] as well as in the curing and causing of disease.

The subject is too large for me to do more than allude to a few things, which seem to apply rather to adults. The daily papers are a most prolific source of disease by means of suggestion, especially the advertisements of nostrums and quacks with their painful detail of symptoms, and the sensational description of disease, whether of given individuals, or taken from hospital lectures, or the like.

But even more baneful than the newspapers is the habit of

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[4] What power suggestion may have in training is well illustrated by an instance recently under my observation. A child, aged 13 years, was brought to me by a probation officer of the juvenile court. The school-teachers could do nothing with him and he was pronounced incorrigible. The officer brought him to me because she had heard of operations being performed on the brains of incorrigibles, and she wished to know if anything could be done with this child. After examining the child carefully, I spoke to him of the satisfaction there was in getting along with teachers and companions, and the joy there was in having the love of all. I then gave him some tablets with instructions to take one three times a day. I assured him they would strengthen his nerves so that he could conquer himself, show the fine qualities that were in him, and gain the love and admiration of all. From that moment he was a changed boy, and there has been no complaint of him since.

making sickness and the sick the common topic of conversation.[5]

Such suggestive influences, as just stated, appear to produce their blighting effects more upon adults than upon children, but they are not altogether without effect on the latter. Children, unfortunately, read the daily papers more than they should be permitted to do, and the subjects of conversation which appear to fall upon their ears unheeded, are often silently and insidiously producing results as pernicious as they are unrecognized.

The influences I have mentioned are hard to combat. They leave no trace on the normal, vigorous individual; and to ward off their ill-effects on the weakly we must aim,—which is the whole purpose of our training,—to build up vigorous health.

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[5] I think it would be a blessing if physicians would take greater pains to impress upon the community the mental tortures often inflicted upon the sick and nervous by speaking much to them about their illness, or about illness in general; and, not rarely, even by the simple question "How do you feel?", which may bring their condition so vividly to their minds. Not less a source of mental torture is the advice so freely given to the sick on every hand, advice to try a given remedy, to change physicians, and the like. The harm is only partly in the fact that the advice may be taken to the patient's detriment. The state of mind produced, indecision, uncertainty, etc., is sometimes lametable. Even the physician scarcely appreciates the loyalty of the patient who has clung to him throughout.

Medicine

PULMONARY ABSCESS ENDING IN RECOVERY WITHOUT OPERATION; A STUDY BY MEANS OF THE RÖNTGEN RAYS [1]

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THE history of the case on which this report is based is as follows:

F. R., aged 28 years, married, an engineer, was admitted to the wards of the Medico-Chirurgical Hospital, November 6, 1905. His family history, previous medical history, and social history showed nothing worthy of record. The present illness for which he was admitted to the hospital began about October 1, with a feeling of languor. This was followed by the usual prodromes of typhoid fever. On October 21 he took to his bed, after a chill of moderate severity; he felt feverish, and perspired occasionally at night. The physical examination on admission showed a white man of average height, well-nourished, with firm, well-developed muscles; the glands were normal; the skin showed the characteristic typhoid eruption. The tongue was heavily coated, white, and moist; the chest was large, of the emphysematous type. The right lung from the sixth rib downward posteriorly showed impaired percussion resonance, slight decrease in tactile fremitus, and a slight decrease in the vocal resonance (thickened pleura). Elsewhere in the lungs sibilant

[1] Read at a meeting of the Section on General Medicine of the College of Physicians of Philadelphia.

and wheezing râles and prolonged expiration were heard. The spleen was enlarged; the abdomen soft, not distended, and nowhere tender. The blood showed 5,023,000 erythrocytes, 4,800 leukocytes, and 85 per cent. of hemoglobin. The Widal reaction was not present in specimens taken November 6 and 10, but was present in specimens of November 11 and subsequently. No malarial parasites were found in the blood. The urine showed the diazo reaction, but otherwise nothing abnormal.

The temperature throughout the course of the typhoid fever was of the intermittent type, each morning falling below the normal and fluctuating between 105° and 97.4° F. (Fig. 1). The pulse-rates varied from 130 on admission to 98; the pulse was dicrotic, but the arteries were not appreciably thickened. No unusual symptoms developed until November 14, when the left leg became swollen and tender (phlebitis). It was wrapped in cotton and elevated, and the symptoms disappeared in about a week.

On November 23, a month after taking to his bed, the temperature rose from 101.4° to 102.4° F., and his cough became worse and was accompanied by the expectoration of rusty-colored sputum. Physical examination showed an area of impaired resonance and broncho-vesicular breathing at the angle of the right scapula. The sputum examination revealed a few pneumococci, but no tubercle bacilli. There were 6,200 leukocytes to the cubic millimeter of blood. The sputum gradually became mucopurulent, but at no time contained tubercle bacilli. The lung improved, but on November 27 there was still some impairment of resonance and harsh breathing in the axillary region.

On December 5 the patient had a severe chill lasting two hours. Dulness, increased vocal resonance, and bronchial breathing were then detected over the entire lower lobe of the right lung posteriorly (consolidation and abscess). The sputum showed the presence of pneumococci.

On December 7, at a point two inches from the midvertebral line on a level with the eighth rib, an area a couple of inches in diameter, of muffled tympany and cavernous breathing was detected.

On December 8, a radiogram (Fig. 2) was made (3 days after the first signs of abscess had developed); this shows an incomplete consolidation of the right lower lobe with a cavity in its upper

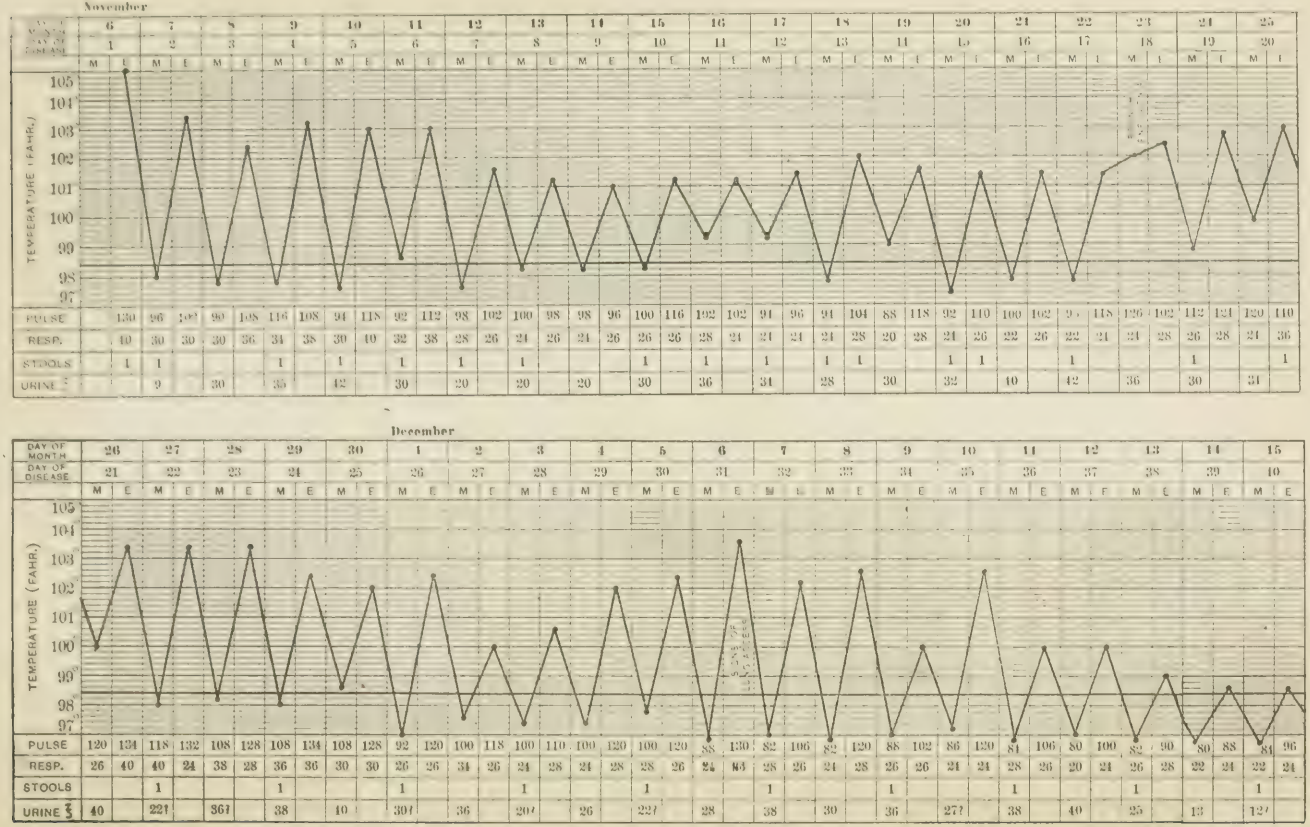

FIG. 1.—Clinical chart of the case of lung abscess.

portion about 2 inches in diameter, extending from the upper border of the fifth rib posteriorly, to the middle of the second interspace, in the midscapular line. The left lung is clear and presents the appearance found in emphysema. There are three enlarged bronchial glands upon the right side (Figs. 1, 2 and 3). The dome of the left side of the diaphragm is resting on a level with the lower border of the tenth rib in the midscapular line, while upon the right side the diaphragm is resting on a level with the lower border of the eighth rib.

On December 9, the blood examination showed a leukocytosis of 10,200, an increase of 4,000 over the number present with simple bronchopneumonia, on November 23. After December 5 (the beginning of signs of abscess) the temperature continued to be irregularly intermittent, the highest point being 103.6° F. The pulse and the respiration-rate were only moderately increased.

On December 11, the patient while sitting up for an examination had a severe paroxysm of coughing and expectorated about two ounces of yellow, purulent sputum, after which he felt better and his breathing improved. Microscopic examination of the sputum showed many pus cells, elastic fibers, a few pneumococci, streptococci, and some staphylococci. The physical signs of beginning resolution in the area of consolidation were noticeable; there was less dulness, while broncho-vesicular breathing and moist râles were heard.

On December 18, the second radiogram was made (Fig.3). It shows less evidence of consolidation in the right lower lobe; the abscess-cavity is still clearly recognized and of the same size. The bronchial glands are less plainly visible and decreased in size. The left side of the diaphragm rests on a level with the tenth rib, while the right remains on a level with the lower border of the eighth rib, in the midscapular lines.

On December 21 the patient seemed brighter and stronger, slept well, did not have night sweats, coughed very little, and only expectorated occasionally. Cavernous breathing was less distinctly heard over the abscess-cavity; percussion gave impaired resonance from the sixth to the eighth ribs in the midscapular line.

On December 29 the breath-sounds in the right lung were gradually approaching to the normal. No distinct cavernous breathing

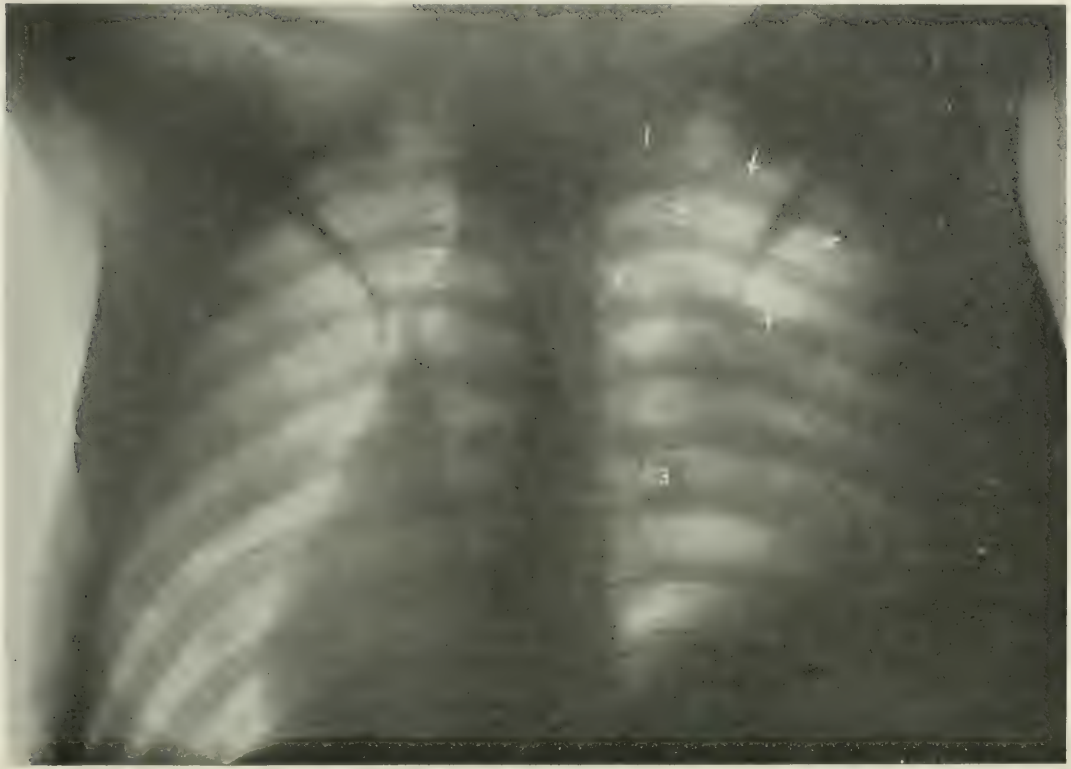

FIG. 2.—Radiogram made on the third day after the beginning of signs of abscess of the lung. The abscess cavity is indicated by arrows. The dark shadow below the arrows indicates the area of consolidation of the lower lobe. The figures 1, 2, and 3 indicate enlarged glands. The diaphragm on the right side is on a level with the lower border of the eighth rib, while on the left side it is on a level with the tenth rib.

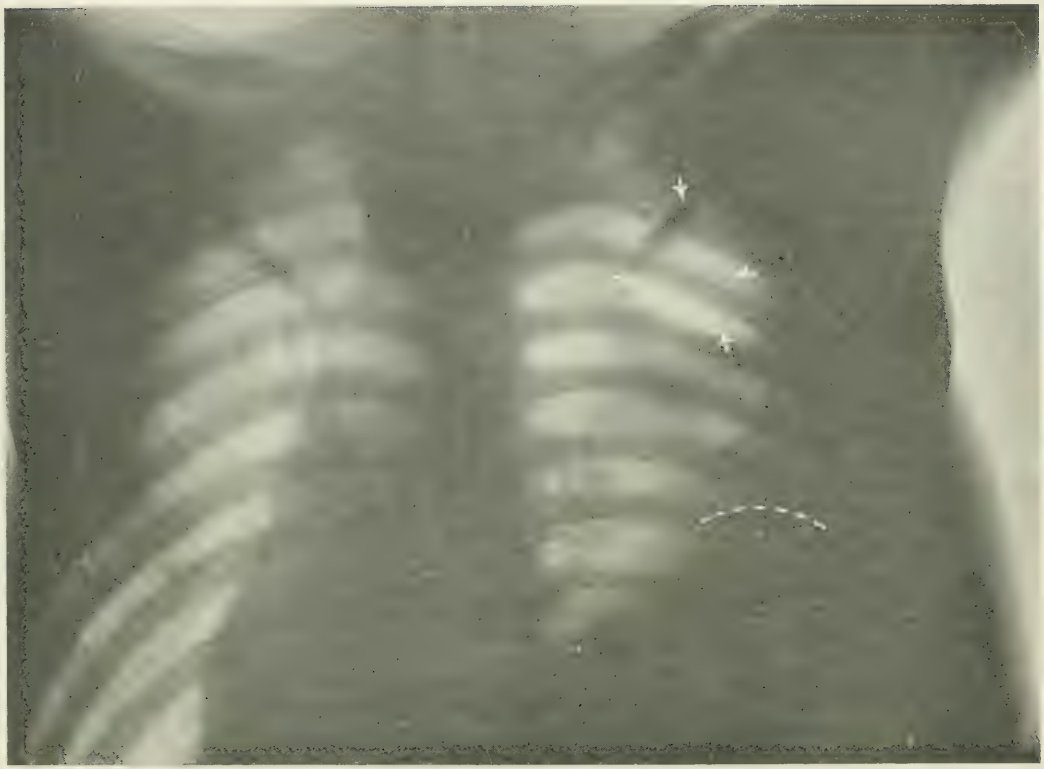

FIG. 3.—Radiogram made ten days after the first radiogram. The abscess cavity and the position of the diaphragm have not changed. The shadow of the area of consolidation is less dense. The enlarged glands are less distinct.

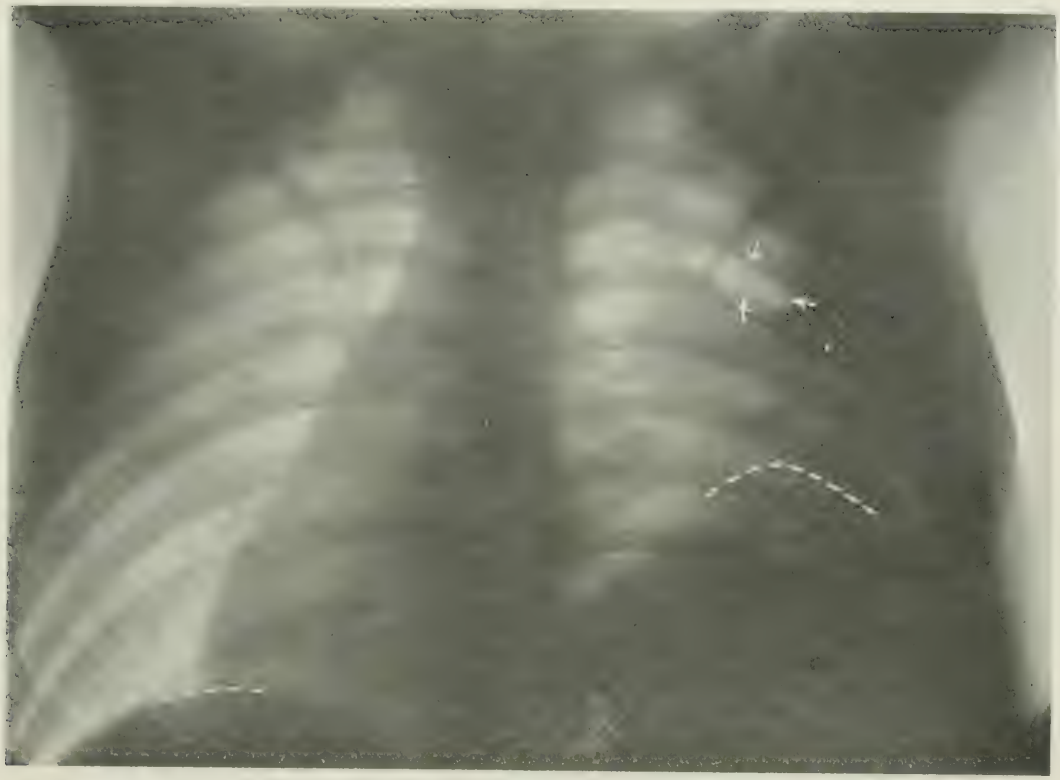

FIG. 4. Radiogram made ten days after the second radiogram. The abscess cavity has decreased about one-half in size. The evidence of consolidation of the lung are less marked. The right half of the diaphragm now is on a level with the lower border of the seventh rib; the left half remains on a level with the tenth rib.

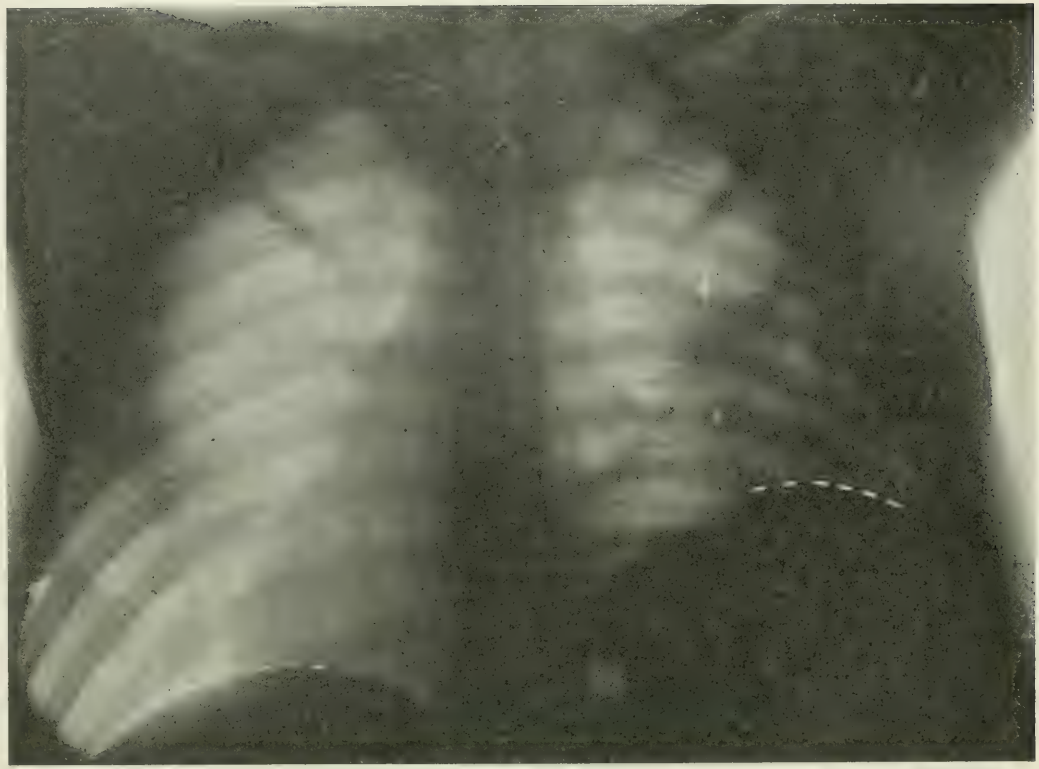

FIG. 5.—Radiogram made ten days after the third radiogram. The abscess cavity is less distinct. The evidences of consolidation of the lung have not changed much. The right half of the diaphragm has receded slightly.

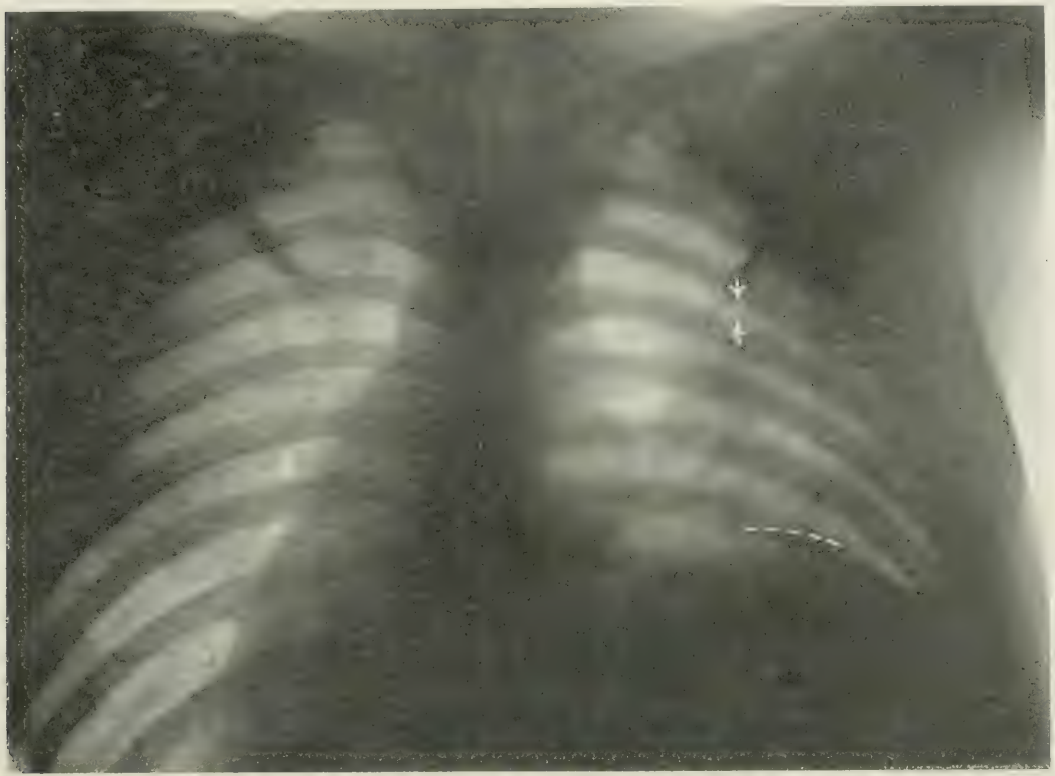

FIG. 6.—Radiogram made ten days after the fourth radiogram. The abscess cavity is reduced to one-half inch in diameter. The shadow of the scar at the lower border of the abscess cavity is apparent. There is still some evidence of consolidation of the lung. The diaphragm has receded to the eighth rib on the right side.

could be detected. The percussion note was still impaired below the sixth rib. The patient was able to sit up in bed. The third radiogram shows less evidence of consolidation of the right lower lobe. The cavity is still visible but has decreased to about one-half its original size. The left side of the diaphragm remains on a level with the tenth rib, while upon the right side it now rises to a level with the lower border of the seventh rib (Fig. 4).

On January 10, the patient had been improving progressively; he spent the entire day out of bed, breathed deeper and more freely, was increasing in strength and weight. Repeated examinations of the sputum failed to show the tubercle bacilli.

The fourth radiogram still shows shadows of remaining consolidation or fibrous-tissue formation in the lower portion of the right lung, with what appears to be a distinct scar below the original site of the abscess-cavity. The position of the diaphragm remains the same as in the previous examination (Fig. 5).

On account of the remains of consolidation in the lung, and the slow tendency to clear up, Röntgen-ray treatment was given during the following ten days. This was suggested by Dr. David L. Edsall before the Section on General Medicine of the College of Physicians of Philadelphia, in 1905, as a result of his investigations upon the increase in metabolism produced by the Röntgen rays.

On December 19, the patient's general condition had been steadily improving. The fifth radiogram (Fig. 6) shows no particular advance in the process of resolution, as a result of the Röntgen-ray treatment. The cavity is now found to be about one-half inch in diameter between the fourth and fifth ribs in the midscapular line. Below this point a distinct scar has formed. The diaphragm has now moved slightly downward upon the right side, and rests on a level with the eighth rib. The left side rests on a level with the eleventh rib. There are no appearances of tubercular deposits in the lungs. No physical signs of cavity can be detected. There are, however, harsh breathing and impaired resonance on a level with the sixth rib in the midscapular line. This is the point at which the shadow of a scar appears to be present.

The patient was discharged from the hospital, to all outward appearances well. He returned to his home in Canada, and further observation has been cut off.

Remarks.—The chief interesting features and complications of this case may be here given *seriatim*. The irregularly intermittent fever-curve without leukocytosis, and all other clinical indications of a suppurative process, up to the date of onset of the intercurrent lobar pneumonia, is worthy of note in the light of subsequent events. The development of phlebitis during the course of the typhoid fever, from which, however, he promptly recovered should be noted. He then developed a pneumococcic bronchopneumonia (November 24), which was apparently clearing up when he had a severe chill, followed by a rapid increase in the area of consolidation, and affecting only the right lower lobe. About the same period, or a little later, he developed the signs of abscess of the lung. This abscess cavity attained its maximum size in about 72 hours. On the sixth day after the beginning of the signs of this condition, the patient had a severe paroxysm of coughing accompanied by free expectoration of purulent material, which seemed to evacuate the abscess. The sputum at this time was found to contain pneumococci, staphylococci, and streptococci as well as elastic fibers. After this fortunate accident the patient improved progressively and rapidly. With the development of intercurrent lobar pneumonia, the temperature assumed the continued type for a few days, when it again became intermittent and so remained until convalescence.

The process was studied by radiograms made at intervals of about ten days. Each radiogram was made by an exposure of six to eight seconds, while the patient held his breath. The patient was in the supine position and the tube centrally placed, at a distance of 18 inches from the plate, on a level with the nipples, and the exposure was made after inspiration.

The diaphragm upon the right side was found not to move downward during inspiration, but rested at the beginning on a level with the lower border of the eighth rib. The third radiogram showed the diaphragm on the right side to be on a level with the lower border of the seventh rib. Therefore the diaphragm in addition to remaining at rest, moved its position upward the distance of the width of one rib and one interspace. This apparently is Nature's process of assisting mechanically in closing an abscess-cavity. In six weeks the abscess cavity was reduced from a

diameter of two inches to a diameter of one-half inch. A heavy scar or possibly a thickened pleura (although no retraction of the chest occurred up to date of discharge) has formed at the lower portion of the original site of the abscess.

The radiograms show that the lower lobe, which was consolidated, has not recovered proportionately with the improvement in the general condition, nor to the degree that the physical signs would indicate. This accords with the observations made by De la Camp [1] in his studies upon pneumonia. He found that remains of the pneumonic process could be found long after the patient had appeared to be well, and after all physical signs of disease had disappeared.

We believe this to be the first case in which the healing process of an abscess of the lung has been studied by a series of radiograms. It illustrates the most accurate method by which such processes can be studied in living man, and presents many advantages over study at the autopsy table. It also illustrates the accuracy with which an abscess of the lung can be localized when an operation seems to be advisable.

It should be stated that surgical treatment was being seriously considered when spontaneous evacuation suddenly occurred, after which the improvement both in the local and general condition was so entirely satisfactory as to remove the necessity for radical measures. We are strongly of the opinion that in pulmonary abscesses occupying a position near to the periphery of the lungs, surgical treatment is ordinarily indicated. In our case the abscess-cavity, as shown by the physical signs, was, comparatively speaking, deep-seated, hence early operative interference was not advised.

Seabury Jones [2] has collected 30 well authenticated cases. Of these 16 were operated on, while 14 were not; of the former 4 died and of the latter 2. We agree with Lewis [3] that in aspiration for diagnostic purposes, the needle may carry infection into healthy tissue. Delatour [4] reports two cases of pulmonary abscess, one of which was secondary to phlebitis of the right iliac vein. H. Gif-

[1] Fortschritte auf dem Gebiete der Röntgenstrahlen, vol. viii, p. 329.
[2] Med. News, 1889, p. 247.
[3] Phila. Med. Jour., 1898, ii, 1368.
[4] Brooklyn Med. Jour., May, 1904.

ford [5] has recorded an interesting case, which occurred in a young man suffering from mastoid disease; this was followed by thrombosis of the lateral sinus. An operation was undertaken and 7 days later he developed signs of lung-affection, while 14 days after the operation the patient expectorated a large quantity of pus, followed in the course of 12 hours by a copious hemorrhage. One day later a second operation was performed with a favorable termination.

Since our patient developed phlebitis affecting the left femoral vein during the attack of typhoid fever, it might be thought that the pulmonary abscess was of embolic origin. But this view is not tenable, since the phlebitis had run its course by November 21, or two weeks before the abscess-formation appeared. Again, embolic abscesses are as a rule multiple, as was true of Delatour's case referred to above, whereas in ours the cavity was single. This condition is believed to be rare as a complication of lobar pneumonia, a view which agrees fully with our experience; but it not uncommonly arises in the course of bronchopneumonia, particularly in the inhalation and deglutition varieties.

Finally, the present case is one of mixed infection and appeared to be clearly secondary to the intercurrent pneumococcic infection of the lung. The treatment embraced nothing of an unusual character.

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[5] Trans. Nebraska State Med. Soc., 1903, p. 76.

THE DIAGNOSIS AND TREATMENT OF ABSCESS AND GANGRENE OF THE LUNGS, WITH SPECIAL REFERENCE TO OPERATION

FROM A STUDY OF THE MASSACHUSETTS GENERAL HOSPITAL CASES

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Difficulty of Diagnosis.—The diagnosis of pulmonary destructive lesions can usually be made with comparatively little trouble. The presence of elastic tissue or fragments of the lung in the sputum is distinctive. In their absence the diagnosis can hardly be made with certainty.

The distinction between abscess and gangrene is especially difficult in clinical cases. It is often uncertain at operation and even at autopsy. Typical examples of the two affections may be easily differentiated, but the two diseases may represent in a single case a beginning as abscess with later the development of gangrene, and it is often difficult to say where the one stops and the other begins. In the presence of putrefactive organisms the pus of an abscess may become as offensive as necrotic pulmonary tissue. In the present paper, therefore, the two diseases will be considered together.

Mistakes on the part of the physician in the number and location of the pulmonary lesions are frequent and at times unavoidable.

In an effort to obtain data bearing on the diagnosis and the surgical treatment of these processes, I have reviewed the autopsy material and the operated cases of abscess and gangrene at the Massachusetts General Hospital. Thirty-eight cases are recorded among the first 1,400 autopsies, and eleven cases have undergone surgical intervention. Bronchiectasis and tubercular processes have been excluded. I am indebted to Dr. J. H. Wright for the use of the autopsy material, and to the surgical staff of the hospital for permission to report their cases.

IMPORTANT DATA FOR SURGICAL INTERFERENCE

The question of operation always arises in the presence of pulmonary abscess or gangrene, and the surgeon may properly look to the physician for the selection of cases suitable for interference. Of especial importance in the consideration of pulmonary surgery are: (1) The number of the lesions, (2) their site, (3) the condition of the pleura, and (4) the presence or absence of pulmonary induration.

(1) The Number of the Lesions.—Having established the presence of a pulmonary destructive process by the finding of elastic tissue or lung fragments in the sputum, the chances are in favor of its multiple character. Thus of 38 cases of abscess or gangrene which have come to autopsy at the hospital, the process was single in 13, multiple in 25.

The relation which the cause of the process bears to the number of the lesions is uncertain and can only be indicated in a general way. Thrombosis, pyemia, bronchopneumonia and aspiration pneumonia seem more likely to lead to multiple foci than to a single focus. Croupous pneumonia was an apparent cause among these 38 autopsies in only 3 cases, twice leading to multiple though circumscribed abscesses. The perforation of subdiaphragmatic suppuration into the lung was followed in one case by a single and in two other cases by multiple pulmonary abscesses.

The duration of the process is also of importance in judging the probable number of the lesions. In general it may be said that the longer pulmonary suppuration has lasted the greater the opportunity for the aspiration of infected material into previously sound parts, causing multiple diffuse lesions, or the extension into contiguous pulmonary tissue with the formation of multiple pockets. This is strikingly indicated in the 11 operated cases. Of 6 patients with acute pulmonary symptoms, the findings at operation and the subsidence of pulmonary symptoms after operation in 5 indicate that the evacuated pus was the only focus, or if other areas existed they were small enough to undergo spontaneous resolution. Among 5 chronic cases, on the contrary, the pulmonary lesions were certainly or probably multiple in 4.

(2) The Site of the Lesions. (*a*) *Involvement of the Different Lobes.*—In the absence of physical signs indicating the loca-

tion of the process, no reliance can be placed on its probable site as a possible point of attack. In this small number of cases, the usual statement concerning the more frequent involvement of the lower lobes is confirmed, but not in a striking manner. The various lobes were affected as follows:

MULTIPLE FOCI

Right	Upper Lobe	8
"	Middle "	6
"	Lower "	13
Left	Upper "	11
"	Lower "	10

SINGLE FOCI

Right	Upper Lobe	4
"	Middle "	1
"	Lower "	4
Left	Upper "	1
"	Lower "	3

(*b*) *Distance from the Pleura.*—On the other hand, the deep or superficial site of the lesions admits of more certain generalization, for of 30 cases in which the position is stated, it is mentioned as just beneath or extending into the pleura in 28. In one case the processes were deep and in another variable, some near and others distant from the pleura. Though in the present series the superficial site of the process is striking, it may vary somewhat with the cause. In the lodgment of foreign bodies in the larger bronchi their erosion may lead to deep-seated processes. In trauma the pulmonary lesions may be found at the seat of the injury.

(3) Involvement of the Pleura.—Pleural invasion may be of value in determining the site of the lesions, from the localization of pain. It may also furnish physical signs of the disease, and when present eliminates one source of danger in operative interference. It is, therefore, of interest to note the frequence of pleural invasion in these cases in which the condition of the pleura is recorded—35. The visceral and parietal layers were firmly adherent in the region of the pulmonary process or over a wider area in 13. The pleuræ were separated by purulent fluid in 5 cases. Weak adhesions or a few bands of connective tissue were found in 12, and the lungs were free in 5.

A consideration of the cause of the process may be of value in judging the probable condition of the pleura. Croupous pneumonia and infection of the lungs from without, either through the thoracic wall or the diaphragm never spare the pleura. In bronchopneumonia or aspiration pneumonia and metastatic lesions, the pleura may be free.

(4) Connective Tissue Formation.—An important distinction must be made between the acute, progressive, and the more chronic lesions. In fresh pulmonary suppuration, the affected regions are not sharply marked out from the surrounding and always inflamed alveolar tissue. In cases which are not progressive, a zone of delimiting granulation tissue appears, and this is later transformed into connective tissue. Spontaneous healing of suppurative foci in the lungs is, however, peculiarly difficult, because of imperfect drainage and the rigidity of the thoracic wall, which prevents shrinkage of the lungs in the closure of a loss of lung substance. The stagnation of pus within the lung is thus readily favored and in turn prevents apposition of the walls of spaces within which it may be confined.

Chronic pulmonary suppuration is always associated with induration, which may be diffuse or confined to the neighborhood of the inflammation. Bronchiectasis and the development of multiple areas from the original focus are frequent. Newly-formed tissue is rich in blood-vessels, the incision or puncture of which may lead to uncontrollable hemorrhage. In older abscess cavities, large vessels may traverse their lumen or line the walls. Erosion of these vessels by extension of the process or mechanical injury may be followed by fatal bleeding. Such cases offer less hope of cure by operation, since the stiff walls of chronic cavities prevent their closure, and the drainage of many though circumscribed foci is practically impossible.

The necessary period for the development of induration can hardly be definitely stated, since it varies in different cases. Marked connective-tissue formation may take place within a few weeks, as in one of the hospital cases—a young man, in whom a large abscess-cavity followed an apparent croupous pneumonia of the left upper lobe. Autopsy three and one-half weeks after the onset of the disease showed that marked induration had already occurred, both about the abscess and in the adjoining part of the left upper

lobe. The operated cases indicate that a longer period may elapse before the case enters a chronic stage, with induration and multiple foci, unfavorable for operation. The cases operated within two months of their onset were usually followed by satisfactory closure of the loss of pulmonary substance, while a delay for many months led to unsatisfactory results. Cases in which the pulmonary suppuration has persisted for years must be only exceptionally suitable for cure by operation. Isolated and striking exceptions, however, are found in the hospital and in the literature.

The clinical aspect of the case, aside from its duration, may afford valuable evidence of the probable condition of the lung. With the continuance of septic features and the persistence in the sputum of elastic tissue and shreds of lung substance, induration is less likely to have occurred to a marked degree. When the suppuration ceases to be progressive, fibrous tissue formation may be assumed to be present and such induration becomes more marked with the progress of time.

DIAGNOSIS

The history of the case often furnishes a clue to the presence of abscess or gangrene. Among suggestive features may be mentioned the occurrence of pulmonary symptoms following operation, especially about the upper respiratory tract or on an individual known to be the subject of bronchial or pulmonary inflammation. An abundant purulent sputum, with septic symptoms after croupous pneumonia, or the presence of suppuration elsewhere in the body may lead to the suspicion of abscess or gangrene. Trauma, empyema breaking through a bronchus, the extension of subdiaphragmatic inflammation, and the inhalation of foreign bodies are usually so striking as to need no special mention. The odor of the sputum is not a safe guide to the presence of abscess. Extensive pulmonary destruction may be present without a foul sputum, and an abundant foul expectoration may occur in bronchiectasis. The sputum is always foul in gangrene, provided the necrotic focus connects with the bronchi.

(1) The Physical Signs of both acute and chronic abscess or gangrene of the lung are notoriously uncertain. The presence of a cavity may add more or less characteristic diagnostic features, such

as amphoric breathing, cracked-pot percussion, bronchial breathing, with a tympanitic percussion note and metallic râles, A variation in the percussion note, with the mouth open and closed, during inspiration and expiration, and on changing the position of the patient, may also be suggestive. Even large cavities may, however, elude careful examination. The smallest cavity, detected as such, during life, in the present series, measured at autopsy 3 cm. in diameter, by 2 cm. deep, and was situated just under the pleura in the posterior aspect of the left lower lobe. Typical signs of cavity are more often present in chronic cases, but for success in operative interference we cannot always safely wait for cavernous signs.

A review of the Massachusetts General Hospital cases illustrates the paucity of distinctive physical signs in acute pulmonary suppuration, even when this is circumscribed and of considerable extent. The persistence of râles at one place in the lungs may suggest the site of a developing process. A friction rub may be heard. Slight variation from the normal in the breathing, voice and fremitus may be present. The cases present little uniformity in such signs, however, in the acute cases. Perhaps the most suggestive of auscultatory signs is the change in the breath sounds in repeated examinations. With the evacuation of the contents of a cavity, the breathing may be distinctly bronchial. A true amphoric quality and metallic râles are seldom present in acute cases. Of the various signs, dulness on percussion over the site of the focus of abscess or gangrene was common to the largest number of cases. It was at times associated with other and confirmatory signs.

In the determination of a single focus of abscess or gangrene and its location, physical examination alone may lead to erroneous conclusions. If reliance is placed only on such evidence, multiple foci may be overlooked. A circumscribed dilatation of the bronchi may be regarded as the site of an abscess or gangrene. In rare instances a circumscribed pleural or interlobular collection of pus may simulate abscess or gangrene. In the exclusion of such sources of error the examination of the sputum is very important.

(2) The Sputum. (*a*) *Macroscopic.*—The gross appearance of the sputum is often suggestive. The sudden expectoration of a large amount of pus may explain previously uncertain symptoms. The appearance of the pus may suggest the number of the pul-

monary foci. In large and single abscesses, or the emptying of outlying suppuration through the bronchi, it is likely to be homogeneous, purulent, and little mixed with mucus. In multiple abscesses, on the contrary, the sputum shows little tendency to confluence, each mass being covered with mucus. Unfortunately an almost pure, purulent sputum, with little admixture of mucus, may occur in advanced diffuse bronchiectasis, with loss of the bronchial mucous membrane. Then, too, an abscess or gangrenous focus may communicate imperfectly, or not at all, with the bronchial tract, and mucus-covered masses of purulent sputum come from inflamed bronchi. A separation of the sputum into three layers on standing has been thought characteristic of abscess or gangrene, but it may be seen in bronchiectasis. A consideration of the history and physical signs may assist in the interpretation of the gross character of the sputum.

(*b*) *Microscopic.*—The presence of elastic tissue or fragments of the lung in the sputum is distinctive of a destructive pulmonary process, provided an alveolar arrangement can be shown. The pleuræ and bronchi also contain elastic fibers, which may rarely cause confusion, unless an alveolar structure can be demonstrated. Without the presence of such elements we can never be perfectly certain of the existence of a pulmonary destructive process. In their absence such lesions may still exist, but unconnected with the bronchi, or in a stage of quiescence. Many of the mistakes in the diagnosis of abscess or gangrene come from a neglect to search for these elements. By physical examination and the microscopic character of the sputum we may establish the presence and location of a destructive lesion. Confirmation of a circumscribed and single process must, however, still be obtained. For the exclusion of other undetected foci, it is essential to examine the lungs with the x-rays.

(3) X-RAYS.—If examination by this means shows a single localized shadow in confirmation of the signs obtained by physical examination and without evidence of other pulmonary foci, we are as certain as our present means of diagnosis permits that multiple areas are absent.

Unfortunately even such a triad of signs, as elastic tissue in the sputum, a circumscribed dulness on percussion, with or without

other confirmatory signs, and a single corresponding shadow with the x-rays, to which Fraenkel[1] especially calls attention, does not exclude the presence of circumscribed multiple lesions, which may be the cause of disappointment at operation.

(4) Exploratory Puncture.—An exploratory needle has often been used in the diagnosis of abscess or gangrene, and frequently with satisfactory results. In other cases, however, it has led to error in diagnosis. In rare cases it is a positive danger. The affected region may be reached, but result in a dry tap, because of the viscid character of the contents. A bronchus containing pus may be entered and an abscess cavity be regarded as the source of the aspirated material. In the puncture of surrounding pneumonic solidification a small focus of abscess or gangrene may escape detection. Whether the procedure is positive or negative, therefore, it is often difficult to interpret its meaning. Its possible danger should not be overlooked.

The withdrawal of an infected needle through an uninfected pleura has led to empyema. Infection of the soft parts may cause a troublesome superficial abscess. Fatal hemorrhage has occurred after puncture of the lung, in a case of abscess of only a few weeks' duration.

PROGNOSIS

The results of abscess or gangrene may be illustrated by: (1) The study of such processes at autopsy on cases of bronchopneumonia or croupous pneumonia (their most frequent cause); (2) the results of clinical observation; and (3) the results of operative interference.

(1) Pathology.—The study of bronchopneumonia and croupous pneumonia at autopsy shows that losses of pulmonary substance are a frequent event. Of 85 cases of bronchopneumonia coming to autopsy at the Massachusetts General Hospital, pulmonary abscesses of various sizes were found either macroscopically or microscopically in 16. In 51 cases of croupous pneumonia, they were present in 14. When the infrequence of permanent pulmonary damage after croupous pneumonia is considered, it is surprising that destructive lesions should be found in so large

[1] Fraenkel, A., Med. Klinik., Berl., 1905, i, 486.

a proportion of such cases at autopsy. Fraenkel,[2] for example, observed a termination of croupous pneumonia in abscess in scarcely 2 per cent. of 1,200 cases. The far greater frequence of abscess at autopsy than among clinical cases suggests that spontaneous healing must take place in a far larger proportion of cases than might otherwise be supposed.

(2) Clinical Cases.—Small multiple or local abscesses are probably frequently overlooked. A severe and prolonged diffuse or localized bronchitis, if examined for elastic fibers, often unexpectedly shows that pulmonary destruction is taking place. I have seen several such cases, in which elastic tissue was found in cases previously regarded as simple local or diffuse bronchitis. Though complete recovery may yet follow, it is in these cases that a final outcome in bronchiectasis, interstitial pneumonia, or chronic abscess is to be feared.

Laënnec[3], Leyden[4], and Wasmer[5] have reported spontaneous recovery in cases with pulmonary gangrene. Of 8 cases of pulmonary gangrene observed by Leyden during two years, 6 were discharged well, one left the hospital with a putrid cavity, and the last was still in the hospital at the date of writing. Wasmer reports 4 cases of abscess and 2 of gangrene, ending in recovery without operation. Elastic tissue or shreds of pulmonary substance were demonstrated in 5 of the cases. A survey of his table shows that resolution appears to have been complete, as judged by the absence of sputum in two, one of whom had had evident signs of cavity, the other of gangrene.

The mortality of abscess, without operation, cannot be accurately indicated. That of gangrene without operation is said by Verneuill[6] to be 80 per cent. Many of the recovered cases have persistent cough and cavity formation.

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[2] Deut. med. Woch., Vereins-Beilage, 1903, p. 204.

[3] Laënnec on the Chest. Trans. from 3rd French Edition, John Forbe, 1838, p. 239.

[4] Volkmann's Klin. Vorträge, 1st series, No. 26, p. 210.

[5] Uber Spontanheilung von Lungenabszesses und Lungengangrän. Inaug.-Diss., Kiel, 1903.

[6] Ann. de la Soc. Belge de Chir., 1900, vol. viii, p. 148. (Quoted from Riesman, Amer. Jour. Med. Sc., 1903, 125.)

(3) Surgical Mortality.—The results of operative interference on cases of abscess or gangrene are given by Garré and Sultan [7] as follows:

Abscess—	96 cases	Cured 77 (80%)	Death 19 (20%)[8]
Gangrene—122 cases		Cured 80 (65%)	Death 42 (34%)

An important difference in the probable success of the operation must be made between the acute and chronic cases, as is strikingly shown in the following 11, from the Massachusetts General Hospital:

Acute Cases.—Six cases of undoubted abscess or gangrene have undergone operation. Their symptoms had lasted for from three weeks to two months, before surgical interference was undertaken. All represent urgent cases, with definite indications for operation in symptoms of sepsis. In 5 of the patients immediate improvement followed evacuation of the pulmonary suppuration. The patients left the hospital in excellent general condition, but with discharging sinuses, to be dressed in the out-patient department or to report later at the hospital. The subsequent fate of 2 of these patients cannot be determined, but in 3 perfect health has been maintained now for four, five, and twelve years respectively after operation. In one case operation was unsuccessful in preventing death, and at autopsy multiple pulmonary abscesses were found.

Chronic Cases.—In striking contrast to the favorable results of operation on acute pulmonary suppuration, is the failure of relief and the high mortality among 5 cases with chronic pulmonary lesions. The duration of the disease varied from nine months to four years before operation. In 4 of the 5 cases, multiple lesions were certainly or probably present. In 3 of the 4, death followed in the hospital, or soon after discharge. In the fourth, three operations in successive years failed to relieve the condition, and the case cannot now be traced. A single successful result is recorded on a patient who suffered from pulmonary symptoms for seventeen months after a pneumonia. The evacuation of four ounces of pus later of some gastric disturbance, but without pulmonary symptoms.

[7] Garré and Sultan. Beiträge zur Klin. Chir., 1902, xxxii, 492.

[8] Such statistics, collected from the literature, lose in value when we consider that a true picture of the operative mortality is obtainable only when all cases of abscess or gangrene, whether favorable or not, have found their way into the literature, a supposition which must at once be discarded.

ACUTE CASES.

Name.	Age.	Cause.	Duration.	Sputum	Haemoptysis.	Abscess or Gangrene.	Site. Single or Multiple.	Operation.	Pleura.	Stay in Hospital.	Condition at Discharge.	Subsequent course.
Jul. R., ...	30	Croupous Pneumonia.	3 weeks.	Not foul.	0	Abscess.	Single R.M.L.(?)	Oiss pus removed; abscess extends 4 inches into lung connects with bronchus.	?	24 days.	Relieved with sinus.	
Olaf A., ...	40	?	2 months.	Foul.	0	Abscess? Gangrene?	Single R. L. L.	Oiss very foul pus-abscess, so large that finger can't reach further side connects with bronchus.	Adh.	24 days.	Much rel'd with sinus. No cough.	Well now, 12 years later
Matthew D., ...	48	Trauma. Immersion in Water.	2 months.	Very foul.	3 IV clotted blood after operation.	Abscess? Gangrene?	Single R. L. L.	Cavity size walnut connects with bronchus.	Adh.?	17 days.	Much rel'd with sinus.	?
Peter S., ...	19	I. Pneumonia II. Empyema	? Probably acute.	Foul.	Frequent, once a pint. In Hospital 5 V at once.	Gangrene.	Single L. L. L.	Pneumonia followed by Empyema: — Drainage. 14 mos. later gangrene lung connects with bronchus.	Empyema.	1 month 3 weeks.	Much rel'd with sinus.	Well now. 5 years later.
Tony C.	60	No history obtained.	6 weeks.	Very foul.	0	Abscess.	Multiple R. L. L.	Evacuation of abscess. Autopsy (1449). Empyema mult. abscesses.	Not adh.	9 days.	Death.	
Kate F., ...	33	Empyema breaking through bronchus.	2 months.	Foul.	0	Abscess.	Single L. L. L.	Simple evacuation of Empyema.	Empyema.	1 month 7 days.	Much rel'd with sinus. Sl. cough.	Well now. 4 years later.

CHRONIC CASES.

Name.	Age.	Cause.	Duration.	Sputum	Hæmoptysis.	Abscess or Gangrene.	Site. Single or Multiple.	Operation.	Pleura.	Stay in Hospital.	Condition at Discharge.	Subsequent course.
Fred'k D., ...	6	Broncho-Pneumonia.	4 years.	Not foul?	Twice in 2 yrs.	Abscess.	Multiple R. L. L.	Lung honeycombed.	Adh.?	3 weeks.	Unrelieved.	Death. 3 weeks later.
C. C. B., ...	18	Pneumonia.	17 months.	Not foul.	0	Abscess.	Single L. L. L.	ʒ IV pus evacuated.	?	1 month.	Well.	Death. 8 years after discharge but without pulmonary symptoms.
Harry B., ...	23	Pneumonia.	9 months.	Very foul.	Frequent, ½ cup to Oi. at a time.	Abscess? Gangrene?	Probably Multiple R. L. L.	I. 1896. evacuation abscess. II. 1897. evacuation abscess. III. 1898. evacuation abscess.	Adh.	In Hosp. 6 times; in all about 8 months.	Slightly relieved each time.	?
Eliz. McC., ...	19	Insidious onset.	11 months.	Foul.	0	Abscess.	Multiple L. L. L.	Evacuation abscess. Autopsy (651) chronic interstitial pneumonia, bronchiectasis, multiple abscesses.	Adh.	8 days.	Death.	
Rob't D., ...	38	Insidious onset.	18 months.	Very foul.	Profuse at times; before entrance 9 hemorrhages in 2 days.	Abscess? Gangrene?	Probably Multiple R. L. L.	ʒ II pus evacuated.	Adh.	3 months.	Unrelieved.	Death soon after discharge

and the patient left the hospital well, to die at home eight years later of some gastric disturbance, but without pulmonary symptoms.

INDICATIONS FOR OPERATION

Acute Cases.—In general, it may be said that cases of acute, single abscess or gangrene are the most favorable for operation.

Gangrene.—The necessity for operation on a single gangrenous focus is usually too obvious to need special mention.

Abscess.—The clinical aspect of the case and the character of the sputum are important guides.

With pronounced and progressive, or even persistent and stationary septic features, there can be no question of the necessity for operation. In patients without septic symptoms and the continued expectoration of a large amount of sputum containing elastic tissue, an incision may hasten resolution. In this class the site of the lesion may be of importance in determining whether operation shall be at once undertaken or the case kept for a time under observation, since spontaneous evacuation is more likely to be complete in the upper than in the lower lobes. Careful observation of the case, with a record of the daily amount of sputum may furnish important data in the estimation of progress. In allowing such cases to continue, there is always the danger of auto-infection of sound parts of the lungs and a transition into a stage of permanent pulmonary damage. If spontaneous cure has not resulted within several weeks, it is usually safer to resort to surgical measures.

Chronic Cases.—The exigencies of the individual case must determine the propriety of surgery in cases in which the disease has lasted for months or years. The chances are much in favor of a multiple, even though circumscribed process, and induration of the affected region may prevent the closure of the abscess cavity. To the uncertainty of cure is added the danger of hemorrhage and the establismment of acute bronchopneumonia or aspiration pneumonia, during narcosis.

A careful history of the case may indicate that the destructive process in question is of more recent origin than might otherwise be supposed, and hence more favorable for operation. In the subacute and chronic cases, I believe that a progressive lesion affords a better chance for cure than one in which the disease has become

quiescent. The continued presence of elastic tissue in the sputum may be of assistance in estimating a progressive character.

Though the hope of cure cannot often be offered patients with chronic abscess cavities, a diminution in the amount of expectoration often follows drainage. Weighing on the one hand the increased risk of such operations, and on the other the discomfort and even danger in the daily flooding of the respiratory tract with a large amount of foul sputum, operation may be advised in the faint hope of cure and a certain assurance of considerable, if only temporary, relief.

CONCLUSIONS

(1) Pulmonary destructive lesions are more often single than multiple, and are usually near the pleural surface. The disease is more likely to be single in acute than in chronic cases.

(2) The diagnosis of a single pulmonary abscess or gangrenous focus is difficult, and at times impossible. The most important data for diagnosis are: (*a*) The presence of elastic tissue in the sputum; (*b*) a circumscribed dulness at some place in the lungs, with or without other physical signs; and (*c*) the exclusion of other foci by x-ray examination.

(3) Spontaneous recovery of even considerable areas of pulmonary destruction may occur.

(4) A striking difference in the results of operation can be shown between the acute and the chronic processes. In the former, cure may be effected in a majority of carefully selected cases, while in the latter though improvement may follow, the results of operation are usually unsatisfactory.

THE TRUE SIGNIFICANCE OF URIC ACID

A PAPER READ AT THE MEETING OF THE MEDICAL SOCIETY OF THE COUNTY OF WESTCHESTER, NEW YORK

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It is my purpose this evening to discuss with you the subject of uric acid, lithemia, lithuria, or that condition which is often designated as superacid condition of the blood. Scientifically speaking, all these terms are more or less inaccurate; at the same time they convey, apparently, a great deal to the mind of the physician and layman. At least they imagine that they do. It is perhaps more so in the case of the layman than the physician. Hence it would be unwise to attempt to eradicate completely these well-known terms.

This subject, so commonly designated uric acid, is one of the most frequent conditions that we are called upon to deal with; it is one of the most important, and one that is very poorly understood, especially from the chemico-physiologic view-point, to say nothing of its practical and clinical aspect. It is absolutely necessary to understand fully the chemico-physiologic importance of uric acid before we can discuss this all-important subject intelligently from its clinical and practical side. If you will kindly bear with a little of the chemico-physiologic detail, a much clearer conception of the conditions which are classed as the uric acid, lithemia, etc., can easily be secured.

Uric acid, as you can readily see by looking at the accompanying Table I, is simply one of the normal end products of proteid oxidation reduction. The first and all-important point to be grasped and fully understood in connection with proteid metabolism is the fact that after the proteid molecule has served its purpose—which is isomeric and mechanical in character—in the various

TABLE I. POSSIBLE PRODUCTS OF OXIDATION OF THE PROTEIDS OR "C H N O S" COMPOUNDS.

Oxidation	Proteid	Oxygen used	Urea	Uric Acid	Kreatinin	Oxalic Acid	Lactic Acid	Glucose	Hippuric Acid	Carbon Dioxide	Water	Sulphuric Acid*
Highest	$C_{72}H_{112}N_{18}O_{22}S$	$154(O)=$	$9(CH_4N_2O)$							$+63(CO_2)$	$+37(H_2O)$	$+(H_2SO_4)$
Super		$151(O)=$	$7(CH_4N_2O)+$	$(C_5H_4N_4O_3)$						$+60(CO_2)$	$+39(H_2O)$	$+(H_2SO_4)$
Normal		$139(O)=$	$4(CH_4N_2O)+$	$(C_5H_4N_4O_3)+$	$2(C_4H_7N_3O)$					$+55(CO_2)$	$+38(H_2O)$	$+(H_2SO_4)$
Uric Acid		$136(O)=$	$2(CH_4N_2O)+$	$2(C_5H_4N_4O_3)+$	$2(C_4H_7N_3O)$					$+52(CO_2)$	$+40(H_2O)$	$+(H_2SO_4)$
Oxalic Acid		$129(O)=$	$2(CH_4N_2O)+$	$2(C_5H_4N_4O_3)+$	$2(C_4H_7N_3O)+$	$7(C_2H_2O_4)$				$+38(CO_2)$	$+33(H_2O)$	$+(H_2SO_4)$
Lactic Acid		$94(O)=$	$2(CH_4N_2O)+$	$2(C_5H_4N_4O_3)+$	$2(C_4H_7N_3O)$		$+7(C_3H_2O_4)$			$+31(CO_2)$	$+19(H_2O)$	$+(H_2SO_4)$
Diabetes		$76(O)=$	$2(CH_4N_2O)+$	$2(C_5H_4N_4O_3)+$	$2(C_4H_7N_3O)$			$+5(C_6H_{12}O_6)$		$+22(CO_2)$	$+10(H_2O)$	$+(H_2SO_4)$
Hippuric Acid		$67(O)=$	$2(CH_4N_2O)+$	$2(C_5H_4N_4O_3)+$	$2(C_4H_7N_3O)$				$+4(C_9N_9NO_3)$	$+21(CO_2)$	$+24(H_2O)$	$+(H_2SO_4)$

* Appears in the urine as a sulphate

TABLE II. SHOWING POSITION OF URIC ACID IN THE SCHEME OF SUB-OXIDATION.

Oxidation	Proteid	Oxyg. used	Urea	Uric Acid	Kreatinin	Xanthin	Hypoxanthin	Carbon Dioxide	Water	Sulphuric Acid*
Normal	$C_{72}H_{112}N_{18}O_{22}S$	$139(O)=$	$4(CH_4N_2O)+$	$(C_5H_4N_4O_3)+$	$2(C_4H_7N_3O)$			$+55(CO_2)$	$+38(H_2O)$	$+(H_2SO_4)$
Uric Acid		$136(O)=$	$2(CH_4N_2O)+$	$2(C_5H_4N_4O_3)+$	$2(C_4H_7N_3O)$			$+52(CO_2)$	$+40(H_2O)$	$+(H_2SO_4)$
Xanthine		$135(O)=$	$2(CH_4N_2O)+$	$(C_5H_4N_4O_3)+$	$2(C_4H_7N_3O)+$	$(C_5H_4N_4O_2)$		$+52(CO_2)+$	$+40(H_2O)+$	(H_2SO_4)
Hypoxanthin		$134(O)=$	$2(CH_4N_2O)+$	$(C_5H_4N_4O_3)+$	$2(C_4H_7N_3O)$		$+(C_5H_4N_4O)$	$+52(CO_2)$	$+40(H_2O)+$	(H_2SO_4)

* Appears in the urine as a sulphate.

fluids and structures of the animal economy, it is finally brought round to some one of the various reducing glands, chief among which are the liver and kidneys. As the fully utilized and inanimate proteid molecule reaches these organs it, together with oxygen, is drawn into the protoplasmic substance of the cells constituting the gland, and in these cells it is broken up by the action of the oxygen upon these complex proteid substances. In the renal glands, for instance, the end products are urea, uric acid, creatinin, carbon dioxide, and water—the sulphuric acid molecule having been lost in the transit of the proteid substance through the protoplasm of the epithelial cells constituting the gastric follicles. All the sulphur contained in the proteid constituents of the food is not discharged, however, as sulphuric acid. A part is eliminated as one of the atomic elements which enter into the molecular structure of taurocholic acid, this acid being only one of the many end-products of proteid oxidation produced in the liver cells and eliminated in the bile. Further than this, the sulphur found in the sulphuric and taurocholic acids does not represent nor account for all the sulphur taken into the system in the proteid elements of the food. Some is eliminated in the various enzymes which constitute our digestive ferments, some is found in the etheral sulphates, etc., but the major part is contained in the two acids above mentioned.

With the sulphur atom eliminated from the sulphur-bearing molecule, we are in a position to take up the final oxidation reduction of the fully utilized and inanimate proteid element as it occurs in the renal cells. The only logical explanation for the production of uric acid is that it is formed normally and solely by a process of oxidation reduction of these fully utilized and partially broken-up proteid substances in the protoplasmic structures which constitute the glandular cells of the kidneys. Therefore, uric acid—as such—never exists in the food-stuffs. It is not found in red meat or white meat, neither is it found in any of the albuminous constituents of the vegetable kingdom. It can be manufactured, however, from the albumin contained in the red or the white meat, or from the albumin contained in milk, eggs, or any animal food. It can also be manufactured from the albumin contained in vegetable substances, but it does not exist primarily in the proteid obtained from either kingdom, any more than the innumerable anilin

dyes and coal-tar derivatives exist—as such—in the native coal tar. Once rid of the idea that uric acid, as such, exists in any of the food substances, the whole subject is greatly simplified.

Please do not misunderstand me when I assume for a moment, for the sake of argument, that the food-stuffs do contain free uric acid, because so soon as it enters the alimentary tract, which is the great laboratory of the system, the uric acid is brought into contact with the free alkalies found therein and would at once be converted into a urate. But as all observers admit that a urate never exists in the blood, this alone precludes the possibility of uric acid reaching the blood by this route. As the blood is a strongly alkaline fluid, even if uric acid did reach this fluid it would, by virtue of the common law of chemistry, be converted into a urate; but since it is admitted by all that the urates do not exist in the blood, this supposition will not hold. Twist it as we may, there is no method known to chemistry by which we can logically and accurately assume that uric acid exists in the blood. At the same time, most writers are constantly stating this as a fact. That the proteid molecule in its isomeric transition through the system often reaches the condition in which it is more easily oxidized into a uric acid molecule than into urea, none can deny; but this is quite a different proposition from the one that declares that uric acid, as such, exists in the blood. An antecedent is not the finished product. That this proteid molecule which precedes the reduction that is followed by an excessive production of uric acid is usually a highly toxic body, none can deny; neither can it be denied that it is the element which is largely, if not wholly, responsible for that wide range of symptoms that belong to the condition so loosely spoken of as the uric acid condition or superacid state of the system.

Accepting this theory of the formation of uric acid—namely, oxidation reduction of a certain isomeric form of the proteid molecule in the renal cells—it is easy to explain its presence and the formation of urates, together with the production of the insoluble phosphate or urate of calcium deposited at various points of the body outside the renal structures, and known to all as the gouty deposit,—the permanent and irremovable deposit being in part, possibly, the insoluble phosphate of calcium. The disturbance of metabolism that is concomitant with this condition results in a vicar-

ious action on the part of the various protoplasmic structures throughout the animal economy, and is especially noticeable in the cartilage cells of the joints. The same vicarious action may occur in any protoplasmic structure. As a natural sequence of this vicarious action and imperfect oxidation reduction, uric acid is produced suddenly and very unexpectedly, as every gouty patient will certify, at various points of the body, but especially in connection with the great toe joints. With this quick formation of uric acid in the cartilage cells we have the characteristic symptoms known as the gouty attack. Following this sudden formation of uric acid, there is produced at this local point urates which are either the insoluble phosphate or urate of calcium crystals, or both. Heintz, and more recently Delépine, have found the acicular crystals so common to the gouty deposit to be composed largely of urate of calcium. Viewed in this light it is highly probable that most of the crystalline deposit in connection with the chalk-stones and gouty deposits is composed of urate of calcium and not phosphate of calcium, as we at one time supposed to be the case. The formation of these insoluble salts destroys the irritation of the uric acid and the attack gradually subsides, because the locally deposited urates are practically insoluble and consequently non-irritating as compared with uric acid. The crystalline deposits are now quickly encapsulated by nature's own reparative powers and remain as permanent deposits, and the symptoms known as the gouty attack entirely disappear.

Going back now to the normal production of uric acid, we find that it is necessary to have uric acid produced in the kidneys. But in the normal state it is rapidly, if not instantaneously, converted into a urate, so soon as it is formed. If this does not occur, we have free uric acid in the urine. At this point we pass from the normal to the pathological or, to be scientifically correct, to the condition which clinically speaking should be named over-production of uric acid.

To understand fully the instantaneous formation of the urate and the necessity for the production of uric acid in the renal cells, we must at the same time follow the so-called alkaline phosphates in their transit through the system. The normal phosphate of

the blood is largely a neutral salt, that found in the food is an alkaline salt, and the one in the urine is an acid salt. The presence of the neutral phosphate of sodium in the urine tends to hold the uric acid in solution, but at the same time it also favors a deposition of the earthy phosphates and thus has a tendency to excite the formation of phosphatic calculi. Therefore, it is absolutely essential to rid the urine of this salt and secure in its place one which will hold the earthy phosphates in solution and destroy the irritating properties of a strong solution of uric acid. This is accomplished as follows: The uric acid, as it is formed and discharged from the renal cells into the lumen of the uriniferous tubules, comes in contact with this neutral phosphate of sodium which is constantly escaping from the blood through the Malpighian tufts into the same tubules, with the development of a urate of sodium and the formation of the acid phosphate of sodium in place of the neutral salt, as expressed in the following formula:

$$C_5H_4N_4O_3 + Na_2HPO_4 = C_5H_3N_4O_3Na + NaH_2PO_4$$

Starting further back with the proteid molecule, we find that it is first attacked by oxygen in the epithelial cells constituting the gastric follicles. At this point a part at least of the proteid constituents lose their sulphur atom and the antecedent to uric acid formation is begun, the complete formula being as follows:

$$C_{72}H_{112}N_{18}O_{22}S + O = C_{72}H_{110}N_{18}O_{22} + H_2SO_4.$$

This desulphurized body is the substance out of which urea, creatinin, uric acid, carbon dioxide, and water are produced in the kidneys, as we have already seen in Table I. Many other nitrogenous bodies are constantly produced, in infinitesimal quantities, by this oxidation reduction that is continually going on in the kidney cells, and they are found in the urine (Table II). The five above mentioned, however, are the only excretory bodies that have as yet become thoroughly practical in the study of proteid metabolism. As our knowledge advances these lesser products of oxidation reduction may become equally valuable, but up to the present time this is not the case.

Coming back again to the stomach, we must take care of this constant outflow of sulphuric acid, which is accomplished in this manner: The constantly secreted sulphuric acid attacks the sodium

chloride contained in the food-stuffs, with the formation of hydrochloric acid and sodium sulphate, according to the following formula:

$$H_2SO_4 + 2(NaCl) = 2(HCl) + Na_2SO_4.$$

In this manner the sulphuric acid is, as it were, instantaneously destroyed as fast as it is formed, being replaced by the hydrochloric acid and the sodium sulphate, both of which are essential for a perfect digestion. The production of sodium sulphate in this manner explains an established fact: that there is a greater output of this salt in the excretions than is taken in, in the food and drink. This is the first rational and practical chemico-physiologic explanation that has been given for the production of hydrochloric acid in the stomach. This theory fits all the facts. All the other theories previously advanced have been connected, largely with unknown quantities, and in many particulars did not harmonize with the facts. But in this there is no deviation from well-known facts and it adheres in every particular to established chemical laws.

After the hydrochloric acid has served its purpose in syntonizing the proteid bodies in the stomach, it must be removed from the alimentary tract because the passage of so large a quantity of this acid through the alimentary canal daily would cause undue irritation. Here again nature with her accurate laws of chemistry comes to our aid. The food contains an abundance of the alkaline phosphate of sodium, $Na_3(PO_4)$. The blood requires a considerable quantity of the neutral phosphate of sodium $Na_3H(PO_4)$, which does not exist in the food. Chemical action helps us out of this apparent discrepancy between supply and demand. The hydrochloric acid which is constantly passing from the stomach to the intestinal canal is brought into contact with this highly alkaline phosphate. When this occurs there is a destruction of the acid, a replacement or restoration of the sodium chloride previously lost in the stomach, thus keeping the intake and output of sodium chloride always equal, which is an established fact, and at the same time there is produced the neutral phosphate of sodium so much needed by the blood,—all of which is briefly expressed in the following chemical equation:

$$2(HCl) + 2(Na_3PO_4) = 2(NaCl) + 2(Na_2HPO_4).$$

Now we have produced the neutral phosphate of sodium which we have already found in abundance in the blood, but not in the foodstuffs, and which is essential also for the formation of the urate of sodium and the production of the acid phosphate of sodium in the uriniferous tubules,—the latter salt being required to give the urine its acidity and to hold the earthy phosphates in solution in the urine. The conversion of the uric acid into a soluble urate at the same time that the earthy phosphates are held in solution prevents the formation of either uric acid or the phosphatic form of calculi. The following somewhat complicated equation covers the whole phenomenon:

$$C_{72}H_{112}N_{18}O_{22}S + O = C_{72}H_{110}N_{18}O_{22} + H_2SO_4 + 2(NaCl) = Na_2SO_4 + 2(HCl) + 2(Na_3PO_4) = 2(NaCl) + 2Na_3HPO_4) + 2(C_5H_4N_4O_3) = 2(C_5H_3N_4O_3Na) + 2(NaH_2PO_4).$$

The uric acid represented in this equation is produced in the cells of the kidney as follows:

$$C_{72}H_{110}N_{18}O_{22} + 135\ (O) = 4(CH_4N_2O) + 2(C_4H_7N_3O) + (C_5H_4N_4O_3) + 55(CO_2) + 38(H_2O).$$

From this analysis of the subject it is readily seen that primarily the formation of uric acid depends upon the law of oxidation reduction of the proteid molecule and not upon the law of synthetic formation. This view is vigorously opposed by some but is fully admitted by the best chemists. That uric acid is necessarily formed normally in the renal cells is unquestionably true because this is the first and only point in the body requiring the use of this acid. In simple over-production it is still formed at this point. In profound disturbances in metabolism it may be formed by the law of vicarious action in any protoplasmic structure, but most commonly observed in the cartilage cells of the great toe joints. Therefore, coming down to the practical aspect of the subject, if we have an over-production of uric acid it is always dependent upon three general principles: (1) A defective oxygen supply; (2) taking into the system more food than can be perfectly oxidized; and (3) disturbances in the action of the nervous mechanism.

All three are more or less closely bound together and cannot be altogether separated in the analysis of this class of cases. Yet in the close analysis of these cases we often find that one or the other

is the predominating etiological factor and must be removed before we can re-establish a perfectly normal output of uric acid.

In the study of health and disease there are a few premises that we must accept. If the individual takes the requisite amount of food, the right kind of food, the right amount and kind of out-of-door exercise, and has nothing to over-tax or worry his nervous system, that individual must be well—that is, if he is born with a reasonably sound tissue foundation. Under such circumstances the individual cannot have any abnormal symptoms. Therefore every deviation from the normal standard is the result of one of three things,—eating too much, taking too little out-of-door exercise, or having some undue nervous strain or tension. If a person eats too much, he takes in more food than he can oxidize; if he does not take sufficient out-of-door exercise he gets imperfect oxidation; and if he has shocks and undue strains upon his nervous mechanism they disturb the circulatory and digestive systems and prevent the free introduction of oxygen, and sub-oxidation follows as a natural consequence. The lesions and symptoms commonly classed as lithemia, rheumatism, diabetes, etc., are all conditions of sub-oxidation, the result of an insufficient supply of oxygen being taken into the system to burn up perfectly the proteids of food. In one individual we get rheumatism, in another lithemia, in another diabetes, or Bright's disease, etc. Why in the one case we have one disease, and in another instance under practically the same conditions we have another disease is a fact for which we have no absolute explanation. Two men will be eating practically the same kind of food, taking the same amount of exercise, and having the same amount of nervous strain and worry: one of the two will have a continuous lithemia, and the other will have rheumatism; and yet another under the same conditions will have diabetes, and still another "Bright's disease"—but they all have a sub-oxidation of the proteids.

How can this be explained? I believe it is due to the action of specific bacteria in the alimentary canal, which cause putrefactive disintegration of the foodstuff and thus produce special toxins which when introduced into the blood cause the specific deviation one way or another. The reason for this belief is largely supported by our knowledge of the action of therapeutic agents. We know that if

we introduce a certain form of alkaloid into the system we get a certain train of symptoms—it disturbs the economy in such a way as to give always very nearly the same symptoms. Take, for instance, morphin—we get a uniform train of symptoms after its administration; the same with belladonna, aconite, quinin, etc. Reasoning by analogy, there is every reason to believe that the introduction of the toxins formed in the alimentary tract is the true explanation for the deviation of the oxidation process one way or another. The pathogenic bacteria when injected into the alimentary canal produce certain poisonous elements, which when introduced into the circulation deviate the oxidation one way or another. Or it may be due to the toxalbumins in the blood developed as the result of the action of the toxins introduced into the blood. There does not seem to be any other rational or logical explanation for the deviation of oxidation reduction.

Assuming this to be a fact, we find that clinical observation bears it out, because we cannot control these conditions unless we observe certain rules that tend to clean up the alimentary canal and put a stop to the putrefactive disintegration of the proteids in the foodstuffs. This accomplished, the patient will get well. If we regulate the diet properly, cause the patient to take the proper amount and kind of out-of-door exercise, and relieve the nervous overstrain, he will get well.

So all these conditions of lithemia and all the various types of suboxidation can be made to get well if we recognize this method of the development of this condition of over-production of uric acid, and look upon uric acid purely as a symptom and not as a condition. Free uric acid in the urine is simply a symptom which tells us that the patient is eating too much, taking too little out-of-door exercise, or has some undue nervous tension of the system which disturbs the physiologic functions—the latter tightening up the blood-vessels and preventing the free utilization of the oxygen and the nutritive pabulum. We have to study each individual case separately and find out which of these conditions is at fault. In this greater city of New York we usually find that all three factors come into play. Men eat too much, exercise too little, and are "driven almost to death from morning until night," and thus keep their nervous systems in a state of constant overstrain. It there-

fore becomes necessary to regulate all of these conditions. Consider their diet, give them proper exercise, alleviate as much as possible the nervous wear and tear brought to bear upon them. The first, or dietetic problem, can easily be regulated; the second is managed less easily, and in many instances the third is a very difficult problem indeed. When, however, the diet is scientifically regulated and the proper exercise is taken the patient is much better able to bear the nervous strain which in many cases cannot be removed.

In the regulation of this class of conditions it is absolutely necessary to take fruit—both fresh and cooked—away from these patients if we are to get the desired results. At this point we usually have the hardest battle with the patient, but in a large practise extending over years my experience has convinced me that we cannot clear up these conditions so long as the patient persists in the use of fruit. Many years ago a patient of mine fell into the hands of the insurance people who examined his urine and found a few hyaline casts. He was denied insurance. A word at this point. Very frequently in these cases of over-production of uric acid, hyaline and granular casts may be found in the urine, and possibly a trace of albumin. That does not necessarily mean, however, that the individual has or will have any renal lesion.

The patient referred to certainly did not look as if he had Bright's disease. He weighed 260 or 270 pounds and was the picture of health. However, this was his mental condition—he thought that he had "Bright's disease" and might not live long. I examined his urine and found a diminished urea output and an enormous over-production of uric acid. I found that he had come from out West where he had led an active life. His business was to manage a branch office here which had not been a success. He pitched into the work with all his might, determined to make it succeed. He secured a house as near as possible to the elevated station so as to lose no time walking to and from his business, worked hard, and was getting no out-of-door exercise but still ate heartily. He soon fell into this condition of over-production of uric acid and the insurance examiner found a few hyaline casts and caused him to infer that he had but a short time to live. His condition was fully explained to him and he was told that he could get thoroughly

well if he wanted to; but I had to battle with that man for weeks before I could get him to give up fruit. Finally he appreciated that he was not doing as I wished him to do, and was not getting well, and he then agreed to follow instructions strictly. Within ten days after he stopped the use of fruit the over-production of uric acid ceased and he got well. That is more than ten years ago and the man is still free from Bright's, and if anything is much better than he was ten years ago. I have taken care of a large number of similar patients during the past decade and have always found them to improve so soon as they followed my directions closely. A few years ago the patient above referred to deviated from the strict rules and again fell into a state in which he had a large over-production of uric acid, and no matter what was done for him the condition of suboxidation continued. I supposed he was obeying the rules previously laid down, but one day he called me up over the telephone and said that he did not know but that he had been eating too many grapes that fall. He had a fine arbor of grapes and was very fond of them, and had partaken of them freely. This had thrown his digestive process out of balance and no matter what was done it would not come into line. The minute he stopped eating the grapes everything swung quickly into line. I have seen that happen over and over again. Just so soon as you put fruit into the alimentary canal it starts up an irritation of the canal which excites the putrefactive disintegration of the proteid elements in the food, deviates the normal process, and excites an over-production of uric acid. It is therefore necessary in such cases to prohibit all fruit; and the cooked fruits are worse than the fresh ones for they contain the same material that the raw fruits do plus a considerable amount of cane sugar, which latter substance the individual does not need. If you introduce into the alimentary canal a lot of extraneous sugar the digestive apparatus has to convert it into glucose before it can be absorbed and assimilated. When this occurs you throw into the system a large amount of easily assimilated carbohydrate material which exhausts the oxygen supply and is naturally followed by incomplete oxidation of the proteid molecule and excessive output of uric acid.

So with people who eat heartily of the vegetable compounds and super-starchy foods—the oxygen supply is exhausted before the

more difficult task of oxidizing the proteid is accomplished. I even exclude potato, not because it is indigestible but because the starch of the potato is probably the most easily digested form of starch that is found, and that is just where the trouble comes. It is so easily assimilated that it uses up the oxygen and there is not enough left to oxidize sufficiently the proteid molecules. I have many patients who if they eat potatoes at the night meal will have an over-production of uric acid the next day. Another reason for eliminating the potato is that most people want to eat it three times a day, and they generally want it fried. I had a patient to whom I had forbidden potatoes and in a year or two he began to want them, and I told him that he could have them two or three times a week, but he said that would not do, he wanted them three times a day and wanted them fried; the result was that he took into his system a great deal more of that kind of material than it had any use for, more than he could properly oxidize, and he at once fell back into a state of suboxidation with over-production of uric acid.

What we want in the condition which is described as starch indigestion is not to augment the digestion of the carbohydrates, as many do, but to cut down the quantity of starch introduced in the alimentary canal and to select those forms of starch which are most slowly digested. The form which is most slowly transmuted and most evenly distributed is the starch of rice and macaroni. To my mind these are the most useful forms of the starch compound that we can use, therefore in my dietary I am in the habit of substituting rice and macaroni for potato. A great many patients do not like either one, but after a while they find that they secure better health by using them. Most people have the idea that macaroni is purely a starch compound, whereas it is made from wheat flour in which the starch has been largely eliminated and the gluten left. It is therefore a highly vegetable proteid compound.

It is usually necessary to add some of the green vegetables to our dietary. The reason for this is that milk, eggs, meat, and the white vegetables have a comparatively low percentage of hemoglobin-forming elements and patients kept too strictly upon them become anemic, as you have all noticed in the bottle-fed or prolonged milk-fed baby. If a child or an adult is kept on an exclusive milk diet for a year or two they become anemic. That is because the milk

does not contain a sufficient amount of hemoglobin-forming material, and there is a tendency for the child or adult to grow progressively anemic. So soon as the digestive system of the child becomes sufficiently strong to utilize fully a wider range of food it is wise to introduce some of the vegetable compounds.

The green vegetables which are the best are lettuce, spinach, green peas, and string beans. To these we may add lima beans, baked beans, and a little asparagus—possibly cauliflower and boiled beets.

No matter what the food is, even though the best, if it disagrees with the individual it should be cut out. In some cases beefsteak, which is the best kind of food, absolutely disagrees. I remember such an instance where I told a lady to use beefsteak and it disagreed, but she could take mutton in any form.

Viewed in this light uric acid becomes not a condition to be treated but simply the symptom which tells what is going on in the system, and if we accept this view we can regulate its output. Every case must be studied individually. We must find out where the defect is and remedy it, and then the individual will get well. I have never seen it fail. The patients sometimes become discouraged because they do not secure the result as quickly as they expect, but where a person has been getting into this condition for years he cannot be brought out of it in weeks nor months; sometimes a year or more may be required, but if you treat the individual for a sufficient length of time upon the principles here outlined he is bound to get well.

I recall a patient who when he first came to my office looked more like a man with a carcinoma than with a simple disturbance in metabolism, but after studying the patient fully I had reason to believe that it was a condition of putrefactive disintegration of the food. I began to treat him on the plan which I have described. I had the battle of my life to keep that man at it, for it was some time before he could see the improvement which he desired and which I saw. I saw the uric acid decrease and the urea rise, but the toxic condition did not disappear as rapidly as the changes in the urine would indicate. The man, however, was finally cured and is to-day in a state of perfect health.

The test by which we determine whether or not the alimentary

canal is doing its work well is the so-called indican test. When the proteid molecule undergoes putrefactive disintegration in the alimentary tract there is produced a substance called indican. This substance, technically known as indoxyl potassium sulphate, is absorbed from the intestinal tract and eliminated through the kidney. When this occurs, if you fill a test tube half full with urine and add an equal quantity of hydrochloric acid and permanganate of potassium, if there is indican present you will get as the first change a purplish coloration. Add some chloroform, shake the test tube vigorously and the indican precipitates as a blue deposit. According to the amount of that deposit you have a high or a low degree of indicanuria. If the blue coloration is less and grows less intense as your treatment progresses, you know that the digestive condition is improving. If it grows darker and more intense, the condition is growing worse. You can make this test in five minutes and thereby know what is going on in the alimentary canal. If there is a large amount, it indicates considerable disturbance; if a small amount, the condition is not so serious.

For the over-production of uric acid, the following test is used. Fill a test tube with urine, boil the upper strata, add 3 or 4 drops of 4 per cent. solution of acetic acid, and if the urine is normal you get no appearance or any sign of uric acid crystals. On the other hand, if there is over-production of uric acid there will be crystals of uric acid formed; and depending upon the amount of uric acid crystals which you get there will be a high or a low percentage of suboxidation. If you find to-day, say 20 crystals and a week from now 40, you will know that the condition is just twice as bad, and vice versa. You can therefore not only regulate the over-production, but you can determine when it is improving.

This test came to me in an interesting way some years ago. One of my colleagues had a child with a severe recurring pain in the ilio-trochanteric region. He had a number of orthopedic surgeons and pediatricians see her, and they made all kinds of diagnoses—hip joint disease, pelvic disease, spinal disease, etc. At last he had me look over her urine. There was only a little urine, and I went over it very carefully, but found nothing abnormal. At last, I took what was left and boiled it and added a drop or two of 4 per cent. solution of acetic acid, and set it aside overnight

to see what would happen. The next morning the test tube was literally full of uric acid crystals. The urine had appeared to be perfectly normal. When the father came around I told him to watch the baby and whenever it had one of these attacks to examine the diaper and see if he could find a little pile of red sand on it. In other words, the attacks indicated to my mind that the child was having an over-production of uric acid from time to time, which gave rise to all the symptoms observed. We changed the method of feeding the child and it got entirely well. That was the way I discovered this very simple and easy manner of determining the over-production of uric acid. In this way we have a complete knowledge of what the digestive organs are doing, and thereby we can, to a large extent, determine how they are performing their functions and whether or not the system is properly utilizing the food. By the above test the over-produced uric acid can be made to appear in a comparatively few hours and thus this test becomes of practical avail. The apparent absence of the over-produced uric acid in a seemingly normal specimen of urine is due to the fact that many urines contain a high percentage of neutral phosphate of sodium, and this produces a condition that will hold the uric acid in solution; but by heating it and adding dilute acetic acid chemical action is excited, the neutral phosphate is changed to the acid phosphate of sodium, and then by letting it stand the crystals are formed.

There are no specific drugs for these conditions. I do not believe that we have any specific drug for such a condition, yet I am a firm believer in drugs and their practical value. No drug is a specific remedy but drugs often greatly assist nature. This is perhaps a very broad statement, but I am very firm in my conviction that not even mercury or the iodides are specifics in the treatment of syphilis, as is ordinarily taught. I believe that mercury and the iodides are very essential in enabling nature to eliminate from the syphilitic individual the toxin which produces the lesions and symptoms that we class as syphilis. I believe that all drugs are poisons or irritants to the protoplasm of the body, and that by virtue of their poisonous or irritating properties they stimulate sluggish protoplasm into action and thus enable the organs to take up the foodstuffs and utilize them. Nature expedites the process not because the mercury is a specific to the poison of syphilis. We are

not justified in making such assertion, because no one has discovered what the specific poison of syphilis is. I believe that it is a toxalbumin, and this is a very difficult substance to isolate. I believe that the action here given is the manner in which the mercury and iodides act as apparent specifics.

The administration of alkalies in connection with this over-production of uric acid will relieve the irritating effects of the uric acid by converting the acid into a urate in the uriniferous tubules, but it does not decrease the over-production of the uric acid. It simply masks a symptom, but never cures. It is sometimes necessary, if an individual is suffering intensely, to administer these alkalies, but by so doing the symptoms are only alleviated. That is what happens to people who go to Carlsbad and various other springs. They come back thinking they are well, but there is just as much over-production of uric acid, only they have a large amount of alkaline fluid passing through the genito-urinary tract which masks the condition; the disturbance in metabolism goes on unchecked, and later the patient often dies unexpectedly just when he is thought to be getting well, because the urine is apparently free from uric acid and micturation is accomplished without pain. Masking of symptoms with therapeutic agents and allowing the disturbances in metabolism to go unchecked is a dangerous procedure.

CONCLUSIONS

(1) Uric acid is one of the normal end products of proteid oxidation reduction.

(2) It is never present in any food substances.

(3) It is never found in the blood.

(4) The antecedent proteid molecule from which uric acid is made is contained in the blood.

(5) Uric acid is made in the renal cells by the oxidation reduction of the desulphurized proteid molecule. The proteid molecule is desulphurized in the epithelial cells of the gastric follicles and in the hepatic cells.

(6) In gout, uric acid is formed in protoplasmic structures other than renal cells by a vicarious action by which the proteid molecule is oxidized at an abnormal point.

(7) It is absolutely necessary to have uric acid produced in the renal cells.

(8) When uric acid is produced faster than it can be converted in the uriniferous tubules into a urate, we have it over-produced.

(9) The urate is produced by the action of the uric acid on the neutral phosphate, thus forming the urate and acid phosphate.

(10) Sulphuric acid is formed in the epithelial cells of the gastric follicles by the splitting off of the sulphur atom from the proteid molecule.

(11) In the cavity of the stomach the sulphur acts upon the sodium chloride, forming hydrochloric acid and sulphate of sodium.

(12) The hydrochloric acid in the lumen of the intestine attacks the alkaline phosphate of sodium, restoring the sodium chloride lost in the stomach and forming the neutral salt for the blood.

(13) The three etiologic factors causing over-production of uric acid are defective oxygen supply, over-feeding, and disturbances in the nervous mechanism.

(14) Uric acid is formed by oxidation reduction and not by synthesis.

(15) The effects of the over-produced uric acid are often confounded with disturbances in metabolism that cause the over-production.

(16) When the right kind and amount of food, and the right kind of exercise are taken, and there is no undue nerve strain, the physiologic economy must work normally.

(17) Different forms of sub-oxidation are the result of the action of specific bacteria acting in the alimentary tract.

(18) Uric acid is simply a symptom and not a condition.

(19) Fruit must be excluded from the dietary.

CLINICAL TYPES OF PERSISTENT VOMITING

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The clinician almost daily must weigh the varied causes of vomiting, and must interpret its equivocal meanings as a symptom. It is necessary to remember that vomiting means discharge of stomach contents and not regurgitation of esophageal accumulation; twice within the past year cases have been referred to me, as stomach affections that proved to be—one a large diverticulum of the esophagus, and the other a spindle-shaped dilatation holding 500 c.c. Attention to the patient's description of how the material is regurgitated and examination of the content will at once determine that there is no true vomiting.

Organic Nervous Disease.—Irregular vomiting, especially if associated with headache, should suggest organic brain disease. In children, pain is often little emphasized, and unmotived vomiting may long be the chief symptom of a brain tumor or tuberculous meningitis. In a child of six years vomiting was a daily feature for five weeks before unequivocal signs of tuberculous meningitis developed. In a girl of ten years, vomiting and polyuria with occasional headache were for weeks the chief symptoms of a specific meningitis. During the past year three cases of cerebrospinal syphilis have been seen wrongly diagnosticated as stomach affections because continued vomiting was the most pronounced complaint. Proper examination of the nervous system at once renders such mistakes impossible.

In some instances repeated vomiting in nervous individuals may be wrongly called hysteric or referred to migraine, or to stomach disorders, until unequivocal signs of organic brain disease develop. One young woman, with a combination of exophthalmic goiter and hysteria, for six months has had frequent irregular vomiting associated lately with headache of moderate severity. Re-

peated examination of the ocular fundus showed nothing abnormal, and the symptom was attributed to thyroid intoxication, until the development within the last two months of optic neuritis and changes in the visual field suggested a tumor of the hypophysis.

Some years ago a man with manifold complaints was seen frequently over a period of two years for attacks of vomiting and headache that seemed accentuations of migraine attacks which he had had all his life: improvement under diet and hydrotherapy further favored such a view, until choked disc and other signs led to the diagnosis of tumor.

A young woman seen two years ago had as chief complaint frequent attacks of vomiting and dizziness that had been variously interpreted as hysteric or due to stomach disturbance. With bladder symptoms and atrophy of the temporal half of the optic discs and absence of abdominal reflexes, there was no difficulty in recognizing the underlying cause, multiple sclerosis. A remarkable case of multiple sclerosis ushered in by incessant vomiting over a period of ten days with vertigo and amaurosis was seen two years ago and classical signs have since made their appearance.

The vomiting attacks of tabes are still not infrequently misinterpreted, and it is not sufficiently understood that crises may not be marked by pain but by vomiting alone. A few months ago a man of 27 years was seen for a supposed stomach affection, as vomiting attacks had occurred at frequent intervals during the past two years; during some attacks pain was entirely absent, though incessant nausea and vomiting tormented him for days. There were typical signs of tabes. As emphasized by Charcot, it is well to be suspicious of the tabetic origin of all periodic vomiting in adults, as gastric crises may be the initial symptom of tabes. A man of 38 years is now under observation who at intervals almost exactly of 30, 90 or 120 days, has extremely severe pain, lasting 36 or 48 hours, in the areas of distribution of the fifth and sixth dorsal nerves on the left side, and coincident incessant vomiting. The only signs are a slight hypalgesia of the affected area, and a sluggish reaction to light of the left pupil: four years ago there was a left hemiplegia which disappeared quickly under specific treatment. The wife of a man of 40 years, who has had periodic vomiting during the past five years, has lately developed cerebrospinal syphilis.

The crises of tabes may be without palpable cause, or may follow a dietetic indiscretion, or may be excited by affections of other organs. A tabetic with gastric crises frequently had storms follow an attack of gall-bladder colic. Senator mentions an instance of cessation of gastric crises for months after nephropexy. Renvers tells of a tabetic with intermittent hydronephrosis and gastric crises, and discusses the therapeutic indications of such sources of irritation.

Von Leyden, in 1882, drew attention to clinical forms of periodic vomiting other than tabetic. The attacks of vomiting may begin suddenly or after slight indigestion, are most often but not always accompanied by violent pain, are associated with obstipation, retraction of the abdomen, and at times with pains in the extremities. In some instances the attacks are much like a severe prolonged migraine paroxysm, in others they resemble a pure stomach affection, and are precepitated by dietetic errors. The vomiting may be incessant for days. In a young woman seen some four years ago there had been migraine at irregular intervals, and her mother and grandmother had migraine. The first severe attack followed chloroform administration, and attacks occurred at intervals of two or three months for two years. The urine was not examined at any of these periods, but administration of sodium bicarbonate has aborted attacks since such treatment was begun. In a man of 40 years whose mother and five sisters had typical migraine, recurrent vomiting seemed to start after a severe stomach attack due to ingestion of bad oysters; there was severe stomach pain, but no headache, with attacks.

Some cases of this kind as well as of periodic hyperchlorhydria may be regarded as migraine equivalents. In a woman of 35 years seen two years ago attacks of migraine and vomiting had become more frequent and severe until finally a state of persistent headache and vomiting was established—the *status hemicranicus* of Moebius or *hemicrania permanens* of Oppenheim.

In a woman of 40 with a history of previous migraine, severe vomiting attacks lasting 4 or 5 days, and accompanied by great prostration and nausea, alternated with attacks of abdominal pain during which large quantities of intestinal sand were passed. In two women recurrent vomiting with at times severe epigastric pain

was associated with angioneurotic edema of the face and extremities.

In all cases of periodic vomiting palpable causes of the paroxysms must not be overlooked. Dietel's crises in movable kidney are well known: intermittent hydronephrosis may long escape detection; the influence of ovarian and uterine disease is notorious; incarceration of inguinal or femoral herniæ must be borne in mind. In an instance of recurrent epigastric pain with persistent vomiting, the presence of an epigastric tumor had led to the diagnosis of gastric carcinoma, but at operation an omental mass, the main content of the epigastric hernia, was removed and relieved all symptoms.

Toxic Vomiting.—The vomiting of nephritis is the best recognized type of toxic vomiting. Some forms of periodic vomiting probably depend upon intoxications not yet identified: the importance of acidosis as an intoxication causing vomiting has been emphasized of late, and there is a growing tendency to regard the vomiting of pregnancy as in great part toxic. Recurrent and most obstinate vomiting may occur in exophthalmic goiter, and is to be interpreted as evidence of thyroid intoxication. This summer a young woman while on vacation at an altitude of 6,000 feet suddenly began to vomit; the vomiting continued incessantly for a week, and was followed by diarrhea, palpitation, and the cardinal signs of Graves's disease. A woman of 55 years was seen some years ago for reputed gastric ulcer as the cause of persistent vomiting, but the condition proved to be an acute exophthalmic goiter.

Gastric Conditions causing Vomiting.—Affections of the stomach as a cause of vomiting are not so likely to be overlooked, and the danger lies rather in accusing the stomach when some reflex, toxic, or nervous influence is at fault. Obstinate vomiting may at times be due to perigastric adhesions and, as the condition is frequently associated with a general nervous state, and often with displacement of the stomach, kidney, and intestines, the diagnosis of the chief factor at work is often most difficult. Separation of the organic part from the functional element can only be accomplished after repeated examinations and careful noting of changes in the stomach outline, with and without distention. In some instances stomachs with adhesions of the smaller curvature of the pylorus to the liver do not show any marked motor insufficiency, but they

are flabby and readily dilatable, they puff out readily on the introduction of even small amounts of air. Not infrequently a pouch near the pylorus will seem to puff out disproportionately as if there were a cause of some local, circumscribed insufficiency of muscles. In a case seen two years ago a large gall-bladder lying across the pylorus was the cause of repeated vomiting attacks. The exaggeration of so-called "reflex" causes of vomiting must be guarded against if misguided therapeutic efforts are to be avoided: there is a tendency to remove the appendix, to suspend the uterus, or to stitch the kidney for the relief of vomiting, when attention to the nervous system would be better therapy.

A number of patients have been seen, chiefly young women, in whom persistent and sometimes incessant vomiting has been largely occasioned by too enthusiastic use of the stomach-tube. The tube has been used wrongly to relieve stomach symptoms that were chiefly nervous—hyperesthesia increases with the persistent use, and finally all articles of food are rejected. Abandonment of the tube, the use of bromides or of bromides with a little chloral, forced feeding, and suggestion usually lead to rapid improvement. In some instances the stomach symptoms may follow immediately a nervous shock or be the sequel of an ordinary gastric upset from dietetic errors. In one young woman sudden loss of appetite and vomiting, which continued for weeks, followed the news of her mother's death by accident.

Recurrent Vomiting in Children.—New interest has been given to this remarkable affection by the valuable paper of Edsall. The vomiting may begin abruptly in apparent perfect health: in a girl of 3 years and a boy of 4 years attacks always began in the night, the children having seemed in usual health on retiring. Prodromes, however, are usually noted—slight indigestion, dulness, nausea, a heavy sweetish odor to the breath, pruritis, a yellowish brown tinge in the complexion. Dr. E. L. Pierson, of Salem, Massachusetts, made the valuable observation that excessive variations or elevations of temperature may precede attacks, and this has lately been noted in one instance. Vomiting may last hours, days or even weeks, and when severe may be accompanied by great prostration, restlessness, or stupor, and not a few fatal cases have been reported. High temperature during an attack is not at all a rare

event. A boy of 9 years seen two years ago had had several attacks each year over a period of six years, with temperature for days of 103°, 104°, and 105° F. A girl of 4 years with attacks of vomiting alternating with intestinal attacks had on several occasions a rectal temperature of 105° and 106° F. Snow and J. P. Crozier Griffith record several cases with high temperatures.

Pain is usually not a constant or even usual feature, but it may be severe, localized to one abdominal quadrant, associated with marked abdominal rigidity, and has several times led to operations for appendicitis or intussusception. In a boy of 11 years seen two years ago, there had been severe abdominal pain with three attacks. The appendix was removed and found nearly normal, but the attacks have been fewer since the operation—probably because they are changing in character and being replaced by migraine.

The odor of acetone is frequently strong in the room in which these children are vomiting, or it is plain on their breath. Occasionally there is a heavy sour or fish-like smell rather than the acetone odor. Snow records analyses of the vomitus, and thinks that some cases probably result from intermittent hypersecretion of highly acid gastric juice. In two cases that I have seen there was no free HCl and a combined acidity of 15 and 20 respectively. Although the association of acetone with digestive disturbances has long been known, and although Marfan in 1901 wrote of a series of cases of vomiting and acetonemia, the credit of associating recurrent vomiting with an acid intoxication belongs certainly to Edsall. Acetone, diacetic acid, and β-oxybutric acid may occur in large amounts in the urine before, at the beginning, and during an attack, and are to be referred to a "cryptogenic acid intoxication"—as yet a mysterious disturbance of metabolism—and not regarded as purely secondary to the digestive disturbance. The frequent brilliant therapeutic results with administration of sodium bicarbonate certainly substantiate the view of a primary acidosis.

Eight cases of this affection have come under my observation since the article of Edsall appeared. In a boy of 9 years there had been severe intestinal indigestion during the first year, and attacks of vomiting began in the second year. Recurrent vomiting lasting 3 to 7, sometimes 10 to 20 days, and accompanied by great prostration and often by high temperature, was the entire com-

plaint. Large amounts of acetone and diacetic acid were found in the urine during one attack and for some days previous to another, and treatment with alkalies has considerably modified late attacks. Of the treatment with sodium bicarbonate the boy's father writes. "It would seem that when we administer the soda it averts the attacks so that they pass away quickly, and are not nearly so intense." A boy of 11 years was perfectly well until he was 18 months old when he had an intestinal attack due to bad milk, with a legacy of intestinal indigestion. Attacks of vomiting began at the age of 4, and have recurred several times each year. They last 24 to 72 hours, are marked by great nausea, prostration, constipation and temperature of 103° to 105° F. Diet and treatment with alkalies modified the attacks considerably, removal of the appendix seemed to affect them still more (as mentioned above), and the last attacks have been preceded by severe headache like migraine; the father and twin sister have migraine. Rachford believes the affection to be closely allied with migraine, and in his cases vomiting disappeared and was replaced by migraine in adolescence. In 3 of my cases migraine was in the family, and the boy mentioned above is now showing headaches with attacks. Two adults with migraine seen the past year give a history of severe recurrent vomiting in childhood.

In a girl of 3 years recurrent vomiting alternated with severe intestinal attacks marked by passage of intestinal sand. At the beginning of either kind of attack acetone and diacetic acid were found in large amounts in the urine, and alkaline treatment has modified both the stomach and intestinal attacks. Edsall records a similar case mentioned by Hamill. A boy of 2 years, brother of the girl above, had recurrent intestinal attacks with fever, with acetone and diacetic acid in large amounts, but no vomiting. The children in my series range from 2 to 11 years. Acetone and diacetic acid (and twice when sought for, β-oxybutyric acid) have been found in the urine at the beginning as well as during attacks, and seem the cause rather than the effect of the disturbance. In two instances oncoming attacks were foretold by the urine examination. Five children have had no severe attacks since the alkaline treatment was begun; in two the severity of attacks has been distinctly modified; one boy has been somewhat benefited but

vomiting still occurs and is now preceded by headache. The sodium bicarbonate is best given, as pointed out by Pierson, before the vomiting has become severe. Snow mentions the interesting fact of a girl of 5 years who craved soda, preferring it to sweets. The parents learn to recognize prodromes of the attack, and are instructed to begin coffee-spoonful doses of the soda every two hours until symptoms disappear. In three children the effect of a few doses of the alkali has been almost magical—vomiting ceasing within two or three hours. In some cases repeated doses will be necessary—8 to 12 grams daily. The urine should be rendered alkaline as soon as possible, and kept so for some days. In two children the soda was not retained, but 5 per cent. soda enemas stopped the attacks in a few hours.

In many of the children nervous influences seem the chief factor in precipitating attacks; the children are high-strung and nervous, there is frequently a history of migraine or functional nervous diseases in the family, and these nervous tendencies must be met by proper therapy. Metabolism of carbohydrates and frequently of fats is faulty, and carbohydrates in the diet must at times be reduced to a minimum. Pancreatic extract has seemed of definite value in two or three instances, and mercury with chalk is another valuable drug. It is not wise to give soda continuously, as indigestion may occur and large doses prove irritating—as in a case of Sharpless. If the alkaline treatment fails to control the vomiting, bromides and chloral by rectum or hypodermics of morphin will be of great value.

During the past year two cases of severe and persistent vomiting have been observed in women of 35 and 40 years, associated with evidence of apparent primary acid intoxication. In one woman "showers" of casts, hyalin, granular, and cellular, appeared in the urine during an attack, and, with the large amounts of acetone and diacetic acid, the specimens resembled greatly the urine seen in cases of diabetic coma. Sodium bicarbonate controlled the vomiting absolutely when morphin, bromide, and chloral had failed. It is important to remember that whenever acidosis is a factor in vomiting every attempt should be made to give food, as starvation tends to increase still further the acid intoxication.

Nervous Vomiting.—There is a type of obstinate vomiting

that can be classed under no more definite heads than "nervous," "hysteric" or "neurasthenic" vomiting.

Some four years ago a woman was seen in the hospital who had been vomiting incessantly for nine weeks; a diagnosis of probable carcinoma had been made, and laparotomy decided upon. The woman of 38 years was emaciated, exhausted, rejecting immediately the simplest food; there was no free HCl in the vomitus; there was some pain in the epigastrium. There was no organic nervous disease, no pregnancy, the urine was normal, stigmata of hysteria were absent; but there was a long history of nerve wear and tear, and a peculiar indifference to the long continued vomiting. Drugs and rectal feeding had been tried in vain. With assurance that vomiting would cease on feeding through a stomach-tube, one was passed and nearly a quart of milk and eggs poured in. Vomiting ceased at once, and recovery was rapid.

Four years ago a woman of 35 years was seen after she had been vomiting for ten weeks. Vomiting had seemed at first dependent on recent pregnancy, but did not cease after emptying the uterus. Despite absence of pain and the rejection of large amounts of bile, a pyloroplasty was done for supposed pyloric spasm. Vomiting continued, emaciation became extreme, temperature rose to 102° and 103° F., stupor deepened, and death seemed imminent. The picture suggested an organic nervous or stomach affection, but there were no objective findings, and there was a strong neurotic taint. A stomach-tube was passed, and a pint of milk and eggs introduced: vomiting did not recur, but convalescence was tedious and marked by a peculiar state of indifference and forgetfulness. The case would speak strongly against the theory of the purely toxic origin of the vomiting of pregnancy.

Some three years ago a young woman was seen who had been vomiting four weeks. There were no physical signs of a stomach affection, characteristic euphoria and indifference were marked, and no drugs had had any effect. Interesting was a history of persistent vomiting during pregnancy some years before that had yielded only after the uterus was emptied. As the period was delayed, curettement was done, but it revealed no pregnancy and vomiting continued. Isolation, chloral, bromides, morphin, etc., did no good, and the heart condition gave cause for much anxiety.

Attempt to pass a stomach-tube proved ineffectual by reason of esophageal spasm. Finally during an endeavor to force the passage of the tube there was a violent hysteric outbreak, and vomiting ceased from that moment.

It is not always easy at the bedside to determine that vomiting which has continued weeks or even months is of purely functional nature. Diagnosis is made by excluding organic causes, by the nervous history and appearance of the patient, by the rather characteristic euphoria and indifference, and perhaps by the presence of other nervous stigmata. The vomiting may occur after the simplest foods, and is often projectile and without effort. There may be strange tolerance of peculiar articles of food, and the strangest procedures may be found to influence, such as giving food in a bath, standing patients on their heads, etc.; the literature of such cases is most entertaining. It cannot be too strongly emphasized, however, that not a few of these patients die. Osler mentions two fatalities, Lowenfeld two, Briquet two, Pomme one, Georget one, and Robinson one.

Treatment by limiting food and choosing diet is a mistake. Isolation and rigid control are necessary. It has seemed wise at times not only to suggest change of nurses, but also change of physicians —the patient's whole surroundings must be radically altered. The much vaunted stomach sedatives do little good in my experience, and electricity has been of no value. Bromides, chloral and morphin are the drugs of which most may be expected. Laparotomy and an operation on the stomach had no influence in one of the above cases; Durand has lately written a thesis on surgical intervention in rebellious vomiting of hysterics.

The affection is chiefly a central nervous one, and must be reached by suggestion or remedies that act upon the center. Varied procedures may thus effect cures, sometimes startling and dramatic. Filling the stomach with a large quantity of milk through a tube led to cessation of vomiting in two of the above cases. The effect may be partly psychical, but in part may be due to distention of the stomach and to influence on possible hysterogenic zones.

From examinations of the urine in two cases, there would seem to be no primary acidosis; in one there was considerable acetone, but probably as result of the long continued vomiting and not as its cause.

Closely allied with this form of nervous vomiting is the incoercible vomiting of pregnancy. Some years ago a young woman of 20 years was seen who had been vomiting some weeks, and was at death's door—there were no external signs of pregnancy, and a pelvic examination had been made with reported negative result. The case was regarded as nervous vomiting, but autopsy showed pregnancy of two months duration. The toxic theory of hyperemesis gravidarum has lately been emphasized anew chiefly by Boehm, Fischel, Lindenman, Dirmoser, Williams, and Ewing, but it would seem wisest not wholly to disregard the nervous element. Toxic influences may be at work in this and the nervous form of vomiting, but they are undoubtedly acting on a nervous system unstable in government of reflexes.

ON THE USE OF DISCARDED STREET CARS IN THE OPEN-AIR TREATMENT OF TUBERCULOSIS [1]

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At the present time there are in many parts of the country old street cars and trolley cars that have been abandoned because of the advent of better equipment. As an example of one excellent use to which these old cars may be put, during the progress of the anti-tuberculous crusade when the amount of money really necessary for the accommodation of patients is not available, I may be allowed to describe what has been done with them at Gabriels Sanatorium near Paul Smith's station in the Adirondacks. About a dozen old horse cars are arranged here in a rather large circle, each of them being fitted up for the accommodation of one patient. Almost needless to say in many ways they present an ideal dwelling for those who must live the out-door life if their pulmonary tuberculosis is not to advance. The experiment of using them has now been in progress for more than two years and they have given excellent satisfaction.

Some of the reasons for this are not far to seek. It has always been felt that the close association of consumptives, even under the strict rules of sanatorium life, may present some dangers of mutual reinfection. We know enough now about the varying virulence of different tubercule bacilli to realize that this might be a source

[1] When this article was written I was not aware that street cars had been used for the open-air treatment of tuberculosis previously to their employment at Gabriels Sanatorium. Since then I have learned that they were first employed at the Pine Ridge Sanatorium near Providence, Rhode Island, at the suggestion of Dr. W. H. Porter, and that Dr. Guy Hinsdale wrote a paper on the subject which was published in the *Colorado Medical Journal* several years ago, and has recently been incorporated in a Brochure on Tuberculosis by Dr. James R. Long, of Virginia.

FIG. 1.– Exterior of a discarded street-car used in the open-air treatment of tuberculosis at Gabriels Sanatorium, New York.

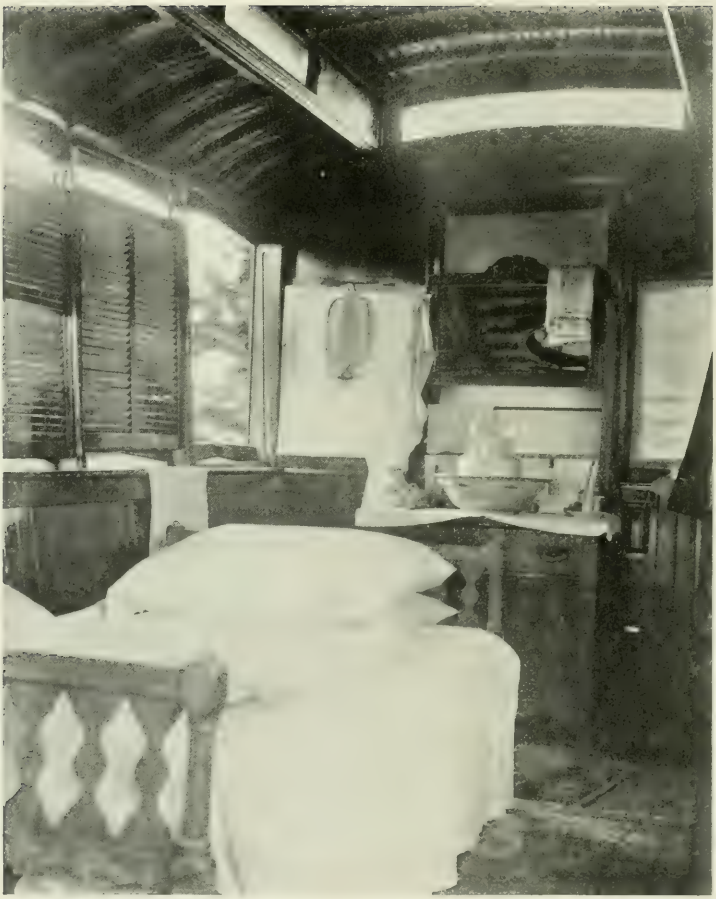

FIG. 2—Interior of a discarded street-car as fitted up for the open-air treatment of tuberculosis at Gabriels Sanatorium, New York.

of real danger especially to patients who have only a very mild form of infection. The tent life with its consequent segregation has been recommended by some for no other reason than this, and the more complete living in the out-door air which it affords. The modern tent, however, as constructed for habitation by tuberculous patients, partakes in all its lower part at least of the nature of a house. It was found that in the old-fashioned canvas tents the draughts along the floor and the tendencies to dampness, even where there was a substantial wood pavement, made life too uncomfortable for the patient. Modern tents resemble the old street cars much more than the old-fashioned tents.

There are other features that make this dwelling in a little habitation by oneself of special significance for tuberculous patients. There is no disturbance from the coughing of neighbors, for the cars can be placed at good distances from one another, and there is no disturbance if a fellow sufferer should be ill during the night and require some special assistance from nurses or the physician. Electric bells can of course be used to summon attendants if necessary quite as if in a building. There is no rude awakening in the morning by an untutored step along the corridor or by some awkward moving in the next room. Besides the patient may decorate his little dwelling to suit himself, and thus find in an occupation of mind a sense of personal satisfaction and a gradual lessening of the homesickness that makes sanatorium life so difficult for so many patients. Nostalgia is, indeed, one of the most serious complications for young patients. Patients may, in addition to this, cultivate the little plot of ground near their car if, as is so often the case, they are to occupy it from the spring until the fall, and thus do away with some of the sense of having nothing to do, which makes time hang heavily on the hands.

The illustrations (Figs. 1 and 2) serve to show very well the exterior and the interior of the cars as they have been fitted up at Gabriels Sanatorium. It was considered better to leave the exterior absolutely as it had been originally, since there was a sense of romance in living in an old horse car and there were many jokes about the road to "Wellville" and "All aboard for Healthtown" connected with the antiquated signs on them. The interior, as may be seen from the pictures, underwent considerable modification.

The seats, of course, were removed and the two sides of the car made more cosy looking and more comfortable by a wainscoting reaching as high as the windows. One door of the car, that toward the north, was permanently closed and this portion was boarded up so as to prevent any of the colder blowing winds of the north from penetrating. Inside the car there is space for one very cosy little sleeping-and-living room.

Almost needless to say these cars in their present condition admit of thorough cleansing and disinfection whenever there is a change of occupant. The hose may be used freely and with the confidence that is begotten of the knowledge that no plaster will be injured above or below and that the sun will thoroughly dry it as soon as it is given a free opportunity to do so. Such old cars may be obtained practically free of charge in many parts of the country, and in the present lively interest in the anti-tuberculous crusade railroad companies may even be found ready to transport them to their destination without charge. They make a very effective means of piecing out accommodations whenever these are inadequate for the number of patients who apply, and yet are by no means merely a makeshift, but constitute a very helpful and almost ideal dwelling during the summer months at least for tuberculous patients.

AN UNUSUALLY LARGE ANEURYSM OF THE ABDOMINAL AORTA

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This case of unusually large aneurysm is deemed worthy of report on account of its etiology, position, enormous size, and resultant nervous symptoms.

H. L., a male, aged 35 years, a teamster, and a native of Germany, came to St. John's Medical Clinic, September 21, 1905, complaining of severe, constant pain in the left lumbar region. He volunteered the information that he had been treated by two or more physicians for over two years for lumbago, but instead of being relieved the pains were growing steadily worse.

His mother is alive, aged 74 years, and in good health. His father died 16 years ago at the age of 55 years from a heart lesion secondary to rheumatism. Three brothers and one sister are well and healthy. The patient has had no serious illness, particularly no rheumatism, and syphilis is denied. He drinks beer, but in other respects his habits are good.

Three years ago he received an injury by a lumber wagon that he was driving colliding with a street car, in consequence of which he was thrown from the wagon and the lumber was overturned upon him. Upon being removed to the City Hospital he was found to be extensively bruised, to have two scalp wounds, and some degree of concussion of the brain. No fracture being found he was sent home the same evening. He remained in bed at his home for two weeks, suffering the greatest pain in the left lumbar region; after sixteen weeks of invalidity he tried to resume his former occupation, but on lifting heavy objects he had pain in the left side, which increased in severity on exertion until he was compelled to give up his work entirely.

About the middle of April, 1905, he noticed over the painful area a swelling which was hard to the touch, but not painful to pressure. He thinks this came on in the night, and that it has

increased in size since then. About the same time he noticed that his left leg began to tremble, and he had pains radiating down the outside of the thigh, from the hip to the knee. During the last few months, he states he has had at different times girdle sensations.

The patient sleeps very little on account of the constant dull, boring pain. Walking or standing for a short time greatly aggravates the pain over the hip bone and outer surface of the left thigh, and in the last three months he says that the peculiar tremble in front of the left thigh has become jumping in character. The jumping is so severe at times when walking that he is compelled to stand still and for several moments hold firmly the muscles, with the left thigh flexed on the abdomen and the leg flexed on the thigh, before he can continue further. When lying down he must be careful to keep the "knee bent" or the jumping will begin. He can rest, as he expresses it, only "curled up," and is not able to sleep on his right side. He has lost about forty pounds in the last eight or nine months. His appetite is fairly good. There is no history of bladder or rectal disturbance.

The patient's expression indicates long suffering and anxiety. A well-marked eruption (iodism) is seen over the face and body. On standing, the patient rests his weight on his right leg, with his body leaning to the right side, and abducts his left leg and foot with his toes resting on the floor, relaxing as much as possible the muscles of that leg. On attempting to sit, he puts his hands on the arms or seat of the chair, cautiously and easily letting himself down, leaning to the right side and continuing to support the body with his arms. His gait is cautious; none too steady; and not spastic. When first examined there was marked clonic contractions of the quadriceps extensor of the left leg alone, but now of the right quadriceps also, together with the glutei adductors and "ham string" muscles of both sides.

When the clothing is removed there is noticed an exaggerated teamster's curve (kyphosis). From a history of his having worn shoulder braces, it is believed he has had this from boyhood; the patient thinks, however, that he has become much more stooped of late. There is also a scoliosis, of which he has been unaware until recently; the upper curve extends from the spine of the fourth dorsal vertebra to the spine of the twelfth; the lower curve from

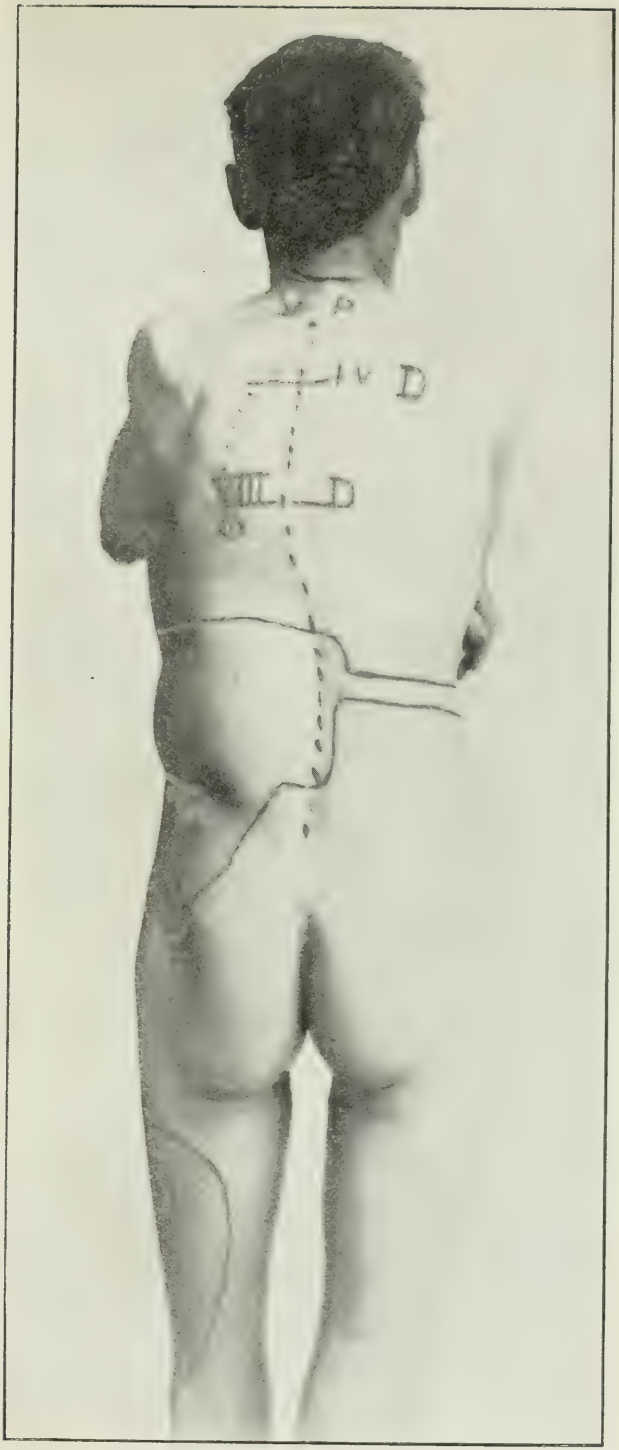

FIG. 1.—V. P., vertebra prominens. IV D. fourth dorsal spine. VIII D. eighth dorsal spine. Dotted line to left of dorsal vertebræ—superior external border of the aneurism. ⊙ Beginning of Traube's relative dullness extending to the ninth intercostal cartilage. Black lines on trunk—hyperesthetic area, upper left side corresponding to the distribution of the seventh spinal segment; lower to left iliohypogastric nerve. On the right, distribution of the eighth spinal segment. On the left thigh, at first hyperesthetic, afterward hypesthetic, area corresponding to the distribution of the external cutaneous femoral nerve.

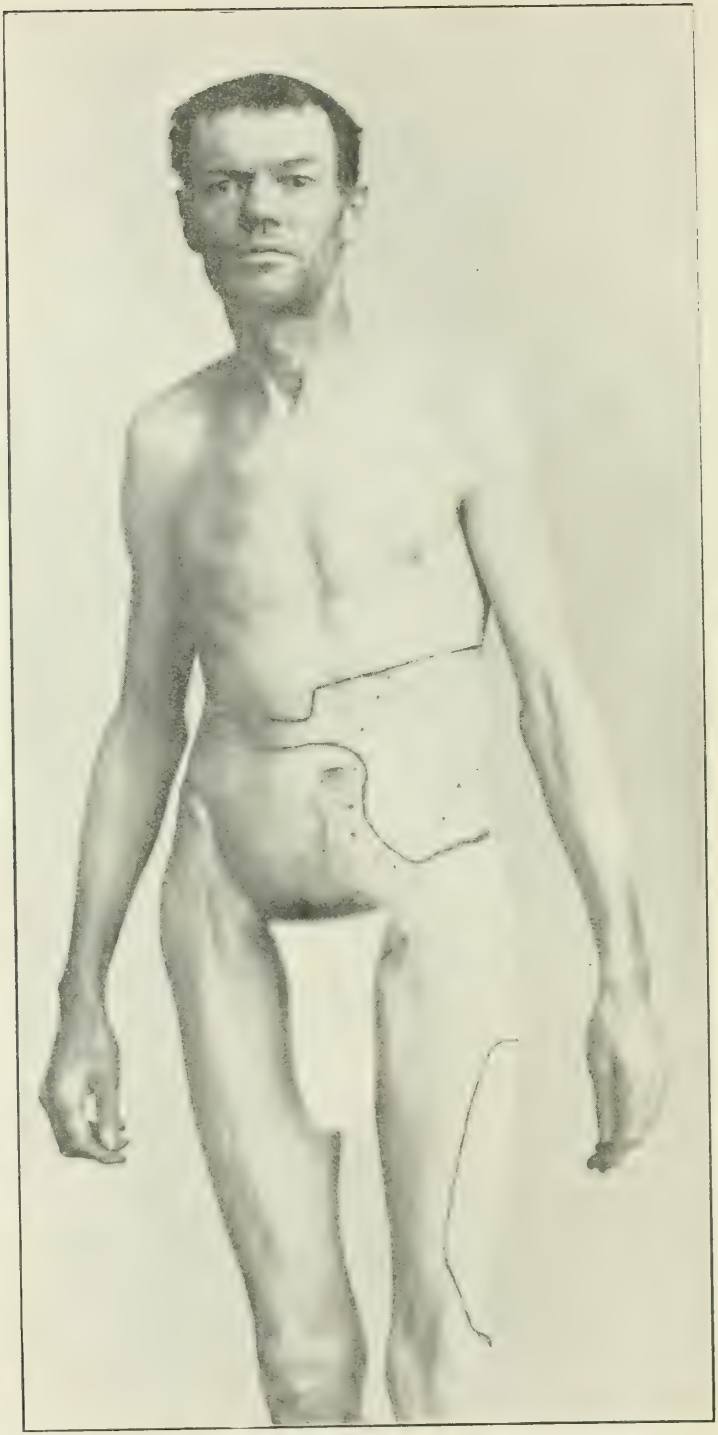

FIG. 2.—Dotted lines, outline of tumor. Black lines, as described in figure 1.

the spine of the twelfth dorsal vertebra to the spine of the first sacral vertebra. In the concavity of the lower curve and to the left of the bodies of the lumbar vertebræ is seen a large pulsating tumor projecting between the bony land marks, and filling the entire space; it is bounded above by the left twelfth rib, to the right by the lumbar vertebræ, and below by the superior border of the crest of the left ilium. In the dorsal region, between the fourth and the twelfth spinous processes, a slight fulness is noticed.

Some enlargement of the left abdominal side is noticed, including the left hypochondriac, umbilical, iliac, and lumbar regions, and this area can be seen to pulsate.

With the right hand over the left lumbar region, and the left hand on the upper iliac or umbilical region, making steady gentle pressure, a tumor of considerable resistance is felt conveying a marked forcible expansile impulse, synchronous with the heart beat; also on the left side of the dorsal vertebra from the fifth down, quite a little resistance is felt. On the right tenth rib is a well marked bony prominence, which more than likely was a fracture sustained by the patient at the time of the injury, and unnoticed until the present time.

The heart sounds are clear, forcible and regular, with the apex beat at the lower fifth interspace in the mammary line. The carotid artery has a peculiar recoil. The femoral arteries can be felt to pulsate on both sides and are very weak, as is likewise the dorsalis pedis.

Posterially, to the left of the spinous process of the third dorsal vertebra, there is dulness of about one finger's breadth; just below, on a line with the fourth dorsal spine, the dulness is two finger's breadth, steadily increasing to the eighth spinous process, where it is four finger's breadth. Below this point there is relative dulness over Traube's area. To the left complete dulness is found over the ninth intercostal cartilage, which can be traced downward just to the left of the umbilicus and linea alba below, to a plain midway between the anterior superior spine of the ilium, and the spine of the pubes. A distinct bruit is heard over the entire dull area, except in Traube's space. Figs. 1 and 2 illustrate the extent of the dull area.

By the physical signs an elliptical-shaped tumor is made out, with its long diameter downward, a little forward, and outward. The long diameter of this tumor is approximately seventeen to eighteen inches, the short diameter about eleven and one-half to twelve inches.

Dr. Graves' report on the examination of the patient's nervous system is as follows:

"The cranial nerves show normal functions. There is a coarse tremor in the slightly closed eyelids, in the protruded tongue, in the lips when the patient shows his teeth and when talking, and in the fingers when separated. McCarthy's reflex and 'jaw jerk' are active. Quinquaud's symptom of phalangeal crepitation is present in the fingers of both hands. The motility of the neck and upper extremities during active and resisted movements is entirely normal. Passive movements reveal no rigidities in these parts. The muscles and accessible nerve trunks of the upper extremities are not tender to pressure. The radial and triceps reflexes are rather exaggerated on both sides, but no difference is apparent between the right and the left. Rombergism is not present. The gait is neither ataxic nor spastic, but it is notably careful, and the trunk is kept in a rather fixed position and inclined rather slightly toward the right as the patient walks to and fro through the room. When standing, the weight of trunk is invariably shifted to the right lower extremity, while the left assumes a position of slight flexion at the hip and knee, with the foot turned outward and resting easily on the toes. When the patient stands firmly on both legs, constant clonic contractions soon become visible in the quadriceps and adductors, and after a few minutes similar contractions may be seen in the 'ham string' and 'calf' muscles of both extremities. Upon grasping lightly the circumference of either thigh with both hands this clonic spasm of the muscles may be felt as rapid and rather uniform contractions.

"The fifth, sixth and seventh dorsal spines are tender to firm pressure, especially the fifth and sixth. The seventh and eighth are rather more prominent than the adjacent spines above and below, and the eighth shows the greatest deviation from the median line. The dorsal kyphosis likewise reaches its greatest height at about the eighth dorsal spine. Coughing or sneezing, or sudden jars, as in walking, are said not to cause pain in the spine; neither does

suddenly applied pressure to the top of the head or to the shoulder provoke pain. When a test-tube containing hot water is slowly drawn downward over the spinous processes the patient cries out with pain when the fifth dorsal spine is reached. The pain thus provoked is said to be equally intense when the hot tube is placed over the sixth and seventh spines. A similar test with a Faradic current of moderate intensity gives similar results. The spine is held in a fixed and rigid position, whether the patient is lying, sitting, or standing. There are no muscle atrophies of the lower extremities, and joint excursions are normal, both to voluntary and resisted movements. There is no difference between the right and the left as regards muscle strength, and the same is in proportion to muscle volume.

"Passive movements reveal a decided spasticity in the quadriceps muscles, less in the calf muscles, and still less in the adductors. The knee jerks are equal but enormously exaggerated; the slightest taps are sufficient to set up 'clonus' in the quadriceps muscle. Patellar clonus is readily obtained on both sides. The Achilles jerks are likewise exaggerated, but less active than the knee jerks. Ankle clonus is present on both sides, but is not decided. Plantar reflex is normal. Babinski's and Oppenheim's reflexes are absent. The abdominal reflexes are present and active in the lower and middle divisions, but in the upper divisions they are very weak, and at times not obtainable.

"A sensory examination of the trunk reveals a zone of hyperesthesia about one inch wide extending 'girdle' fashion about the trunk and corresponding approximately to the skin distribution of the seventh dorsal segment on the left, and the eighth dorsal segment on the right. This zone corresponds accurately to the location of the girdle sensation, of which the patient has complained from time to time. Sensation is nowhere diminished below this zone. On the right the lower margin of this zone can be as accurately determined as the upper margin. On the left, the upper margin of this zone becomes united latterly and posteriorly with, and cannot be separated from, the upper margin of a hyperesthetic area, which corresponds roughly to the outlines of the tumor. Still another hyperesthetic area is found on the outer surface of the left thigh, corresponding closely to the distribution of the external cutaneous

femoral nerve. The hyperesthetic zones and areas are outlined on the patient's body and are best studied by inspection. Within the areas thus outlined hyperesthesia is present to pinpricks, temperature, and touch.

" The sensory findings just read were obtained November 8, and on the morning of November 9, the patient states that he had suffered no pain in left leg during the night. In going over the sensory examination again, the findings were the same as on the 8th, excepting within the distribution of the external cutaneous femoral nerve, where pinpricks were scarcely perceived as such. Touch was not perceived as acutely as in the adjacent skin, but temperature hyperesthesia was still present. A third sensory examination, made November 10, revealed the findings to be practically the same as on the 9th. The patient has not had a recurrence of pain about the outer surface of left thigh.

"A consideration of the objective findings alone afford ample evidence upon which a diagnosis of both central and peripheral involvement of the nervous system may be made.

" The objective findings indicating involvement of the spinal cord are:

" (1) The zone hyperesthesia extending about the trunk corresponding to the segmental distribution of the seventh dorsal nerve on the left, and the eighth dorsal nerve on the right.

" (2) Decided and visible clonic spasm of the muscles of the lower extremities when the patient is standing.

" (3) Exaggeration of knee jerks and Achilles' reflections, with patellar clonus and ankle clonus.

" (4) The decided spasticity of quadriceps, ' calf,' and adductor muscles, as elicited by passive movements.

" (5) Tenderness to pressure over the fifth, sixth, and seventh dorsal spines, and the pain provoked by the application of heat or Faradic current to these spines.

" (6) The decided weakness and failure at times of the upper abdominal reflexes.

" The upper level of the cord lesion probably corresponds to the seventh dorsal segment and should therefore be found opposite the fifth dorsal spine. The lesion up to the present moment is undoubtedly slight in character—probably slight pressure—since all signs are absent indicating a severer lesion.

"The objective findings indicating a disturbance in the peripheral nerves are:

"(1) A sharply defined area of hyperesthesia, corresponding to the distribution of the left ilio-hypogastric nerve.

"(2) A similar area corresponding to the destruction of the left external cutaneous femoral nerve.

"(3) Absence of the cremasteric reflex on the left.

"These areas of hyperesthesia and absence of the cremasteric reflex on the left, are probably due to direct and gradually increasing pressure on certain nerves by the lower part of the tumor. The assumption of such pressure finds justification in the striking change from hyperesthesia to hypesthesia in the external cutaneous femoral nerve, which occurred during the night between the 8th and 9th of November."

Subsequent sensory examinations were made on November 18 and 21, and showed a decidedly increased hyperesthesia of the inner side of the thigh with general hyperesthesia of the leg and foot, while the area corresponding to the distribution of the external cutaneous femoral nerve seemed to be a little more hypesthetic than on November 9 and 10.

On November 24, at 1 A.M., the patient called his nurse, complaining of a most severe pain in the upper left lumbar region, and remarked, that he was of the opinion, something had ruptured inside of him. This pain continued until death occurred, about forty-five minutes later.

An autopsy was performed November 24, 1905, at 3 o'clock P.M., at St. John's Hospital. The following notes were made: Rigor mortis is pronounced. The pupils are equal and in a moderate state of dilatation. The skin is profoundly anemic. The visible mucus membrane are very pale. No foreign bodies are found in mouth or nasal passages, nor are there any signs of violence or bruises on the body. The fatty tissue is scanty in amount, while the bony and muscular tissues are fairly well developed. A pronounced bulging is present in the left lumbar region manifesting itself principally posteriorly and moderately laterally, extending from the free margin of the ribs to the brim of the pelvis. This mass is circumscribed and easily compressible. The kypho-scoliosis of the spinal column is well marked. The spinal cord is of uniform consistency throughout, except at a level corresponding to the upper

border of the body of the fifth dorsal vertebra. Here the cord is softer and slightly flattened for about one-half an inch. On the anterior wall of the spinal foramen at the upper border of the body of the fifth dorsal vertebra an eminence projecting backward into the canal can be felt; it is due to a very acute angle in the column at this point.

On making the long anterior median incision, the subcutaneous tissues over the abdominal wall are seen to be filled with a peculiar glazed fluid, undoubtedly aqueous in nature, as though there were a beginning subcutaneous edema. The cut section appeared very pale. Height of the diaphragm: left side, upper border of the fourth rib; right side, lower border of the fourth rib. The cavity of the thorax shows neither fluid or air on either side. The lungs do not approximate in the median line by two inches. The apex of the left lung is bound down by firm adhesions, while the right shows no adhesions. Sections of both lungs reveal a slight amount of fluid mixed with blood and air. The bronchial mucosa is pale. There are several enlarged, calcified, and indurated peribronchial lymph nodes. The pericardial sac contains about a drachm of pale yellowish green serum, but no thickening of the visceral or parietal layers. The heart is slightly hypertrophied and very pale. The right auricle and ventricle and the left auricle are empty. The left ventrical contains a slight amount of red blood clot. The auriculoventricular opening admits three fingers. The mitral valve admits two fingers easily. The aortic valves are competent as regards the water test. The coronary arteries appear normal and empty. No clots are seen in the pulmonary vessels. The intima of the ascending aorta shows a few yellowish plaques, evidently indicating fatty changes. No distinct evidences of arteriosclerosis are found. The abdominal cavity shows a small amount of free sanguinous fluid, and the stomach and intestines slightly distended with gas and very pale. The liver is seen to extend one finger's breadth below the free costal border. The gall ducts are patulous and contain a very small amount of greenish bile. The appendix vermiformis has several adhesions, but is otherwise normal. The liver, spleen, pancreas, stomach, intestines, and right kidney, appear normal, the spleen probably being a little under size. The left kidney is so encapsulated in the large mass that it is deemed unnecessary to examine it since all the other organs are normal. A large tumor mass

is found in the left hypochondriac and lumbar regions, and careful examination shows it to be an aneurysmal sac coming from the abdominal aorta just below the diaphragm and just above the celiac axis. The opening into the sack easily admits two fingers. This sack protruded upward, pushing against the diaphragm, backward, pushing against the last two dorsal and the lumbar vertebræ, downward, to the brim of the pelvis, and forward, pushing the left kidney from its normal position and jamming it firmly against the diaphragm. Besides this large aneurysmal sac there is a distinct hematoma on the left side just in front of the middle of the sac. This envelops the left kidney and is bound down tightly by the peritoneum in front of the left kidney,—that is, it is a retroperitoneal hematoma. Examination shows that this saculated aneurysm of the abdominal aorta had ruptured into the left retroperitoneal space, causing a fatal hemorrhage. This readily accounts for the slow death after the rupture of so large a vessel. The sac is filled with organized blood clots. The aorta above and below the opening into the aneurysmal sac is normal. Both renal arteries and veins are normal. There is marked erosion especially of the twelfth dorsal and first lumbar vertebræ and to a less degree of the second and third lumbar vertebræ.

Aneurysm of the abdominal aorta is comparatively rare, especially one that has attained such enormous size; in fact, I have never seen a report of any case in which an aneurysm has grown to such magnitude.

The etiologic factor in this case is undoubtedly trauma. The history of a grave injury and the succeeding symptoms, together with the absence of history and of symptoms of syphilis, tuberculosis, rheumatism, and arteriosclerosis, confirmed by the autopsy, seem sufficient proof of the truth of this statement.

This aneurysm is believed to be a direct traumatic one in contradistinction to an indirect traumatic one, not only on account of the foregoing reasons, but also on account of the variety of the aneurysm, that is, saculated and wholly on the left side, with the opening into the sac corresponding exactly with the external injury of the trunk.

The resultant nervous symptoms were interesting as well as instructive, especially the latter, for it demonstrates the fact, that irritations of the cord can be located accurately even if they be slight.

CARDIAC LESIONS ASSOCIATED WITH ARTERIOSCLEROSIS

A CLINICAL LECTURE DELIVERED AT THE HOSPITAL COLLEGE OF MEDICINE, LOUISVILLE.

BY FRANK C. WILSON, M.D.

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GENTLEMEN: The patient that I have the pleasure of showing you to-day is a colored man, N. M., aged 70 years, who comes to us complaining of dyspnea, especially, however, of what he calls smothering sensations which have lasted now for a year and which are more marked in the early morning before arising than at any other time. He also has a cough which annoys him especially at night. He has no edema, and states that he has never suffered with rheumatism. He tells us that his parents lived to an old age, and that one brother died as the result of a cough which had persisted for a considerable time; beyond this, however, no information as to his family history can be elicited—as is common in colored subjects.

You will remember the patient as having been before the class several weeks ago—upon which occasion we found a well marked arcus senilis, atheroma of the arteries, and the following physical signs referable to his heart. Over the precordium one could then, and still can, hear a distinct "rushing" murmur, occurring with the first sound of the heart, and heard with maximum intensity over the mitral area near the apex of the heart. A systolic murmur with its point of maximum intensity at this region can scarcely result from anything but regurgitation of the blood through the mitral orifice in consequence of incompetent mitral valves. Futhermore, this murmur is propagated to the left (toward the axilla) and can be heard posteriorly—additional evidence that it is a mitral regurgitant murmur. We may say, then, that there is an insufficiency, or incompetency, of the mitral valves, allowing the regurgitation of blood, and that this is the cause of this murmur.

There is, in addition, an abnormal sound, or murmur, at the base of the heart (to the right of the sternum) which is heard most distinctly at the point denominated the aortic area, and which we know must be produced at the aortic orifice. This murmur is divisible into two parts; the first occurs at the same time as the murmur heard at the apex, that is, when the ventricles are contracting. Such a systolic murmur at the aortic area is usually attributed to obstruction at the aortic orifice, though it may also be due to other causes, such as atheroma, aneurysm, etc. Aortic obstruction, however, does not account for the entire murmur heard in our patient in this location, since the murmur does not cease when the ventricles have finished contracting, but is prolonged into the diastole of the ventricle—the second portion of the murmur alluded to. We know that normally the contraction of the ventricle and the exit of the blood therefrom and its passage along the aorta is followed immediately by dilatation of the ventricle and closure of the aortic valves; normally, then, these valves are closed when the second portion of the murmur in our patient is produced. When, however, the aortic valves do not properly close, regurgitation of the blood from the aorta into the left ventricle occurs, and a diastolic murmur is produced—as in our patient. Furthermore, we find upon listening over the carotid arteries that the first or systolic murmur is well propagated into these vessels—confirmatory evidence that this portion of the murmur is due to aortic obstruction; and the second portion of the murmur can be heard well down toward the ensiform cartilage—confirmatory evidence that it is due to aortic regurgitation. This, then, is what is called a double or "see-saw" aortic murmur. In our patient, therefore, we find three murmurs, a mitral regurgitant, an aortic obstructive, and an aortic regurgitant.

The important question for consideration is, What is the probable cause of these murmurs? We cannot elicit any history of previous rheumatism; we are told that as far back as he can remember he never had rheumatism, and we cannot, therefore, ascribe the organic changes to that cause. To what, then, are we to attribute them? It does not follow with certainty that because a patient gives no history of rheumatism there has not been an attack of rheumatic inflammation; there may be, for instance, serious endocardial inflammation without outward manifestation of rheu-

matism. Not infrequently we encounter patients in whom serious organic heart disease exists and who give no evidence of past or present rheumatism. If our patient exhibited no evidence of atheromatous degeneration, considering the advanced age of the patient, we might reasonably ascribe the organic changes in his heart to so-called latent rheumatism involving primarily the endocardial membrane; but I am not inclined to invoke this cause since the atheromatous degeneration which the patient exhibits to a noteworthy degree fully explains the situation. We know that atheroma may involve the cardiac valves, the endocardium which is reflected over the valves, and also the aorta, as well as the walls of the blood vessels, and that degenerative changes may occur in the walls of the arch of the aorta as well as the various ramifications of other blood vessels throughout the circulatory system. Quite often atheromatous degeneration, as in this patient, involves the radial artery, and it may involve any or all the arteries of the body; other evidence can be seen in the eyes, the arcus senilis.

The cough which this patient has may be secondary to interference with circulation through the lung. When there is interference with the heart action, when the heart cannot propel the blood through the pulmonary circulation with its customary degree of vigor and force, when there is regurgitation through the mitral valve damming up the blood in the pulmonary circulation, when the vigor and force of the right ventricle is impaired by degenerative changes, when there is interference with the nutritive supply of the muscular tissue of the heart through degenerative changes in the coronary arteries,—then congestion of the pulmonary circulation, an outflow of serum, and an alteration in character of the secretion from the pulmonary mucous membrane necessarily result, and cough naturally follows. This man says his cough is most annoying in the latter part of the night, that there is an accumulation of the secretion, and he coughs to clear this away, that the cough does not trouble him much during the day, all of which confirms the idea that it is due to pulmonary congestion.

The next question that arises is, What can we do for our patient? Considering the atheroma present, and the loss of vitality (which is really a part of his advanced age), what is indicated in the way of treatment looking toward betterment of his condition?

The loss of vitality must be combatted by tonics, and among these strychnin is perhaps the most efficient in giving tone and vigor to the heart action. Potassium iodide in moderate doses will fulfil several indications. When congestion of the pulmonary circulation exists and causes the outpouring of serum and exudative material, infiltrating the lung tissue and the bronchial mucous membranes, potassium iodide is distinctly indicated. It is an agent which will stimulate the absorbents and help to clear away exudative and inflammatory material. Chronic bronchial inflammation from any cause is an indication for administrating moderate doses of potassium iodid. The iron tonics are useful, and as this patient is more or less anemic iron will serve to enrich the blood. Anything which will tone up, invigorate, and improve his general condition will have a favorable influence. Therefore, the administration of strychnin with some form of iron, and moderate doses of potassium iodide, will be appropriate. About all we can hope to do is to tone up and invigorate the general system so that the tendency to degenerative change and lower vitality may be corrected as far as possible. Nux vomica is an excellent gastro-intestinal tonic and may be substituted for strychnin. Nearly always in cases of arteriosclerosis involving the smaller arteries there is put upon the heart much extra work or strain in order to force the blood through the more distant portions of the arterial system and this eventually leads to hypertrophy of the left ventricle. The left ventricle oftentimes becomes decidedly hypertrophied since the heart, receiving no assistance from the elasticity of the arteries, has unaided to force the blood through the entire systemic circulation. Ordinarily, when the arteries have their full elasticity, the walls stretch and then rebound under pressure, the vessels are dilated by the first force of the cardiac contraction, and the blood current is shoved along, so to speak, and there is a reinforcement of the power of the heart, a continuation of the force of the heart contraction, which greatly facilitates the circulation.

Hypertrophy of the left ventricle is confirmatory of what we feel in the arteries—calcareous degeneration. So long as nature succeeds in preserving normal equilibrium we may say the condition is one of compensatory hypertrophy. So long as the heart sends blood in sufficient quantity, so long as perfect adjustment is pre-

served, the patient remains fairly comfortable, and the various organic functions are carried on in the natural way, but when nature fails in this the kidneys are among the first organs to suffer. So long as nature succeeds in sending the proper amount of blood through the kidney circulation, excretion goes on normally, but when renal circulation is insufficient to induce proper secretion, failure of the kidney becomes threatened. This is one evidence of the inability of the heart to accomplish its work. When such a condition exists a preparation of digitalis should be added to the strychnin or nux vomica. A form in which I have found this drug very serviceable in such cases is amorphous digitalin (Merck's German) administered in doses of $^1/_5$ to $^1/_2$ grain (0.012 to 0.03 gram). This dose is much larger than is usually given, the maximum dose given by most physicians being about $^1/_{32}$ to $^1/_{16}$ grain (0.002 to 0.004 gram). Yet in cases of this kind from $^1/_5$ to $^1/_2$ grain may be given with beneficial results and without danger. The beneficial action of this preparation of digitalis is greater than that of any other form of the drug in cases of this class. It increases the force and vigor of the circulation, making it more even, and assists the kidneys in a manner which other preparations do not. Strophanthus is also a remedy which, when there are respiratory difficulties, seems to exert a more favorable influence than almost any other heart tonic known. Sulphate of spartein also may be used alone or in combination with other remedies. It should be given in $^1/_4$ grain (0.015 gram) doses every four to six hours, as may be indicated by the conditions present.

For the patient before us I believe the following combination will fulfil the indications, for the present at least: Nux vomica 15 drops (1 c. c.), sulphate of spartein $^1/_4$ grain (0.015 gram), and amorphous digitalin (Merck's German) $^1/_5$ grain (0.012 gram).

Another remedy which is perhaps quite essential in the treatment, is one of the nitrites. Nitroglycerin or any nitrite that will dilate the capillaries and relieve pressure, will be of benefit. If the small blood vessels (the peripheral arterial system) be dilated, the blood is forced through that portion of the circulatory system with less resistance—an essential in the management of a case of this kind.

There is one other condition sometimes observed as a result of arterial degeneration which should be mentioned: When the arch of the aorta is involved, the strength of the walls being thereby weakened, internal pressure may so dilate the vessel as to cause slight rupture, thus producing an aneurysm. Sometimes there is uniform weakening of the walls, and a general enlargement takes place, which is known as dilatation of the arch of the aorta. In such instances a decided pulsation can be detected over the ascending aortic arch and a murmur occurs at that point. The dilatation can be mapped out by percussion, there being an increased area of dulness over the crest of the arch. In the normal subject the dulness which can be made out by percussion in this region is less than one inch in width and can be traced across behind the sternum in the course which the aorta takes. When a greater area of dulness exists in this locality, in connection with the other symptoms described, we may conclude that there is dilatation of the arch of the aorta.

The murmur which can be heard under these circumstances is not infrequently due to calcareous deposits upon the inner surface of the lining membrane of the arch of the aorta; calcareous threads are deposited, giving a roughened and uneven surface instead of the glistening, smooth appearance which it naturally presents.

I have no doubt that this condition of affairs is present in the patient before us since there is marked pulsation and enlargement over the arch of the aorta, certainly more than would be expected in the natural condition of that vessel.

MISCELLANY FROM THE PARIS MEDICAL WORLD: THE USE OF X-RAYS AS A SOCIAL QUESTION; HEROINOMANIA; THE SERUM TREATMENT OF EXOPHTHALMIC GOITER, AND OF HEMOPHILIA

BY CECIL KENT AUSTIN, M.D.

Of Paris, France

THE USE OF X-RAYS AS A SOCIAL QUESTION

When the early period of experimentation with x-rays had passed, it was seen that not only the superficial tissues but deep-seated viscera were affected by this new agent. It was thus found that intense reactions could be produced thereby in the retina, liver, spleen, thymus, spinal marrow, and nerves of the heart, without these reactions having any relation or proportion to that of the superficial tissues.

These facts did not appear to constitute a danger either to patient or physician, so long as the exposure remained within limits incapable of irritating the skin. But recent discoveries seem to show that although this may be true of individuals, it is not true as regards the race, as it has been found that x-rays cause sterility; *they produce sterility in males and females with a facility and safety that can only be looked on as an actual social danger,* to which it seems urgent to call physicians' attention.

Passing over the experiments that have shown that in this way small animals of various kinds can be rendered sterile without the slightest injury to their general condition, skin, sexual desire or vigor, we find that a similar effect can easily be obtained in men and women. Through simply being in the vicinity of an x-ray apparatus during radioscopy or radiotherapy, men have become sterile for a period of time that has not yet been definitely determined, but that may extend over several months. At the end of this time there is a gradual return to the normal condition, and living spermatozoa are finally discovered in the testicular fluid.

This action of radiation is just as evident on the ovary as on

the testicles, though in this case we have only the proof furnished by animals. It has been noted that the ovaries appear more sensitive to x-rays than the abdominal epidermis, which in these experiments showed no trace of irritation. The effect of the Röntgen rays may go so far as atrophy of both testicle and ovary, though whether this is final or not has not yet been elucidated; degeneration is more serious in the case of the ovary than of the testicle, and requires shorter seances.

The first inference from these facts is the pressing necessity of protection of the body, and in particular of the genital zones, in the case of physician and patient during the use of x-ray apparatus. The second is that this discovery is sure to open a vast field of action for unscrupulous charlatans.

When we consider that this method of producing sterility is painless and of easy application, leaves no trace, is accompanied by no cutaneous reaction, and is so effective that a few sittings only suffice to obtain a full result, and if again we consider that in quantities necessary to create a condition of total azoospermia, there is no effect on the general system, no fatigue, no prostration; and when, finally, we consider that the method does not diminish to the slightest degree the patient's virility,—we are warranted in concluding that it is far the best Malthusian agent known.

Although it is probable that x-rays can produce permanent atrophy of the ovary and testicle, it appears to have been shown, at any rate for the testicle, that to accomplish this a large amount of the agent is requisite; so that it would not be difficult at all to produce at will a temporary condition only of azoospermia.

A moment's consideration of this action of the Röntgen rays will convince every one of the urgent necessity of putting under proper control all applications of the x-rays to man. The law prescribes severe penalties for persons causing abortion, and for the patient that seeks it; but in the present state of our legislation any non-medical person resorting to this new practice can do so with impunity.

Up to the present time anyone with the means requisite has been able to acquire the apparatus and use it, and it is to be feared that unless the law intervenes no great period will elapse before more or less shady offices are opened where under pretext of radio-

scopic examinations the two sexes will be reduced to a relative degree of sterility on a large and remunerative scale.

The question then must imperatively be surrounded by the necessary safeguards, the first of which, in view of the real social danger involved, will evidently be that every person not a physician in good and regular standing will be forbidden under serious penalties to use the x-rays in any form on his fellow-beings, male or female.

HEROINOMANIA

It was thought at one time that the morphin habit could be cured by the substitution of cocain; but the only result of this experiment was to create a new habit, that of morphin-cocain, and for a while these were the only cases obtained, a pure case of morphin habit having become a rarity. By the time that this mistake was fully realized, this one, probably worse, arose; for several years, now, physicians have tried to replace morphin by heroin, and at the present day we see nearly as many cases of the heroin habit as of the morphin habit.

There was a semblance of an idea in trying cocain, as the two substances were chemically different; though in fact all that was done was to substitute one poison for another. But with heroin this is not so, as it is a product of the same chemical series,—almost identical, to tell the truth. And in using it even the method of administration is the same, whereas it is desirable that this should be different, in applying the substituted method to the breaking up of a drug-habit.

Enough cases of heroinomania have now been observed to make it possible to denounce the danger this remedy presents, as well as the mistaken ideas people have of it and of the intoxication it produces.

It is a gross error to say that heroin is not more toxic than codein; it is certainly more toxic than morphin at equal doses, and nothing is more striking with heroin-habit patients than their cyanosis, their deficient hematosis, and their very great prostration.

The best proof of this greater toxicity is the shorter time that patients can stand it. They can take large doses of morphin for years, but with heroin they last a far briefer period, even with smaller doses.

People fancy that they gain in the fact that at the start half the

amount of heroin only is required as of morphin to maintain their equilibrium. But as less pleasure is derived from heroin than from morphin, they soon increase their doses to as much or more as they were taking of morphin, by which time they have practically doubled their intoxication. For this reason it is common to see heroin patients taking very large amounts of the drug.

Heroin was first proposed as a substitute in mild cases of the morphin habit; but neither in mild nor severe ones should it ever be used. Not only are such cases not cured thereby, but the patients are exposed to an intoxication more serious than their former one, since the ruin of the system is much more rapid and complete than with morphin.

Heroin seems to have a peculiar action on the brain. With morphin, people often appear for a long time to be in possession of brilliant intellectual qualities; but the effect of heroin is just the contrary, producing mental torpor.

Morphin acts specially on the brain, liver and glands, but not on the circulation and respiration; whereas these are just the functions affected by heroin, particularly the latter. Hematosis is very imperfect, and it is not uncommon to see suffocating attacks of bulbar origin with the heroin habit, even at the beginning. Physiologically heroin seems to act on the medulla oblongata, and morphin on the brain, and for this reason the former has a more serious effect on the vital functions of the body.

The convalescence after the heroin habit is slower, and is accompanied by a much inferior and less satisfactory reaction than in the case of morphin.

The suppression of the drug in a case of heroin habit is not only as difficult as with morphin, but it is far more dangerous. With the rapid (not sudden) method of stopping the use of morphin, if there is cardiac syncope, or alarming weakness, a dose of morphin the tenth part of the final dose used, will suffice to pick up the patient. With heroin this is not true; even a good dose is not enough, and we have to add ether, caffein, etc., which even then may prove insufficient.

In stopping morphin, attacks of syncope are due to heart fatigue, and can be avoided; but with heroin there is no means of avoiding them, and they are a real danger, being respiratory (not cardiac)

and, what is more grave, of bulbar origin. Without any warning the patient begins to breathe with difficulty, cyanosis sets in, breathing stops and the pulse becomes thready; a dose of heroin fails to produce the stimulation one gets from morphin in the morphin habit, and if the patient cannot be revived by other means he will die.

The only way to get around this danger, when one attempts to cure the heroin habit, is to transfer it first back to a morphin habit! When one has made a morphomaniac again out of his patient who had gone over to the heroin habit, he can proceed to cure him of his morphin in the usual way, without being exposed to any such disasters as with the heroin.

Heroin has no superiority to morphin, and many points of inferiority, and could probably be struck out of the pharmacopia without loss.

And now there is another habit on the horizon,—the dionin habit. Cases have already been published, and we shall doubtless have trouble with this drug too in the near future.

SERUMTHERAPY OF EXOPHTHALMIC GOITER

A THEORY was propounded in 1894 that Graves's disease was the result of exaggerated activity of the thyroid gland, and that the more important symptoms of the disorder were produced by an excess of its internal secretion. This made exophthalmic goiter the exact opposite of myxedema, which we know to be due to deficient secretion on the part of the same gland. Consequently, if we could combine the humors of a patient of each of these two categories, the mixture would probably be something approximate to the humors of a normal person.

This was the idea that led to the first attempts in 1895, at a serum treatment of the disease. The thyroid was removed from some animals, and then their serum was taken and administered to a series of patients by Ballet and Enriquez. In general terms there was improvement in all of these cases; the goiter, trembling, and tachycardia were favorably influenced, the most stubborn symptom being the exophthalmos. But these early experiments gave reason for thinking that it would be preferable not to use dog serum, and at the same time that better results might ensue from administering the preparation by the mouth instead of by hypodermic injection.

The next attempts were made by Lanz, who used the milk of goats from which the thyroid had previously been removed, and by Mœbius, who employed sheep serum from animals treated in the same way, but to which he added a little carbolic acid. These methods are, however, only variations on the original one.

The experience of all who have used the method seems to indicate that the hypodermic means is unnecessary; the administration by the mouth of these preparations is just as advantageous and is much less troublesome. Again, it appears that of all the substances tested, the milk and the blood alone of such animals have the desirable therapeutic efficacy.

Now the use of milk is not very practical; it keeps badly, and the attempts made to preserve it for any length of time appear to alter its composition and give it a bad taste. So that the habit has now become general of using the blood only.

Instead of Moebius's carbolized serum, Hallion has thought it preferable to use blood just as it is, combined with glycerin; and to this preparation he has given the name hematothyroidin. Since the serum is active, the blood must be active also, as it contains the serum. But what shows that in the blood the serum is the only efficacious element, or even the most efficacious, in the treatment of this disorder? We know quite well that the leukocytes are the special vehicles of many active substances; why should they not be in the present instance? It is quite possible that this specific substance is to be found principally in the formed elements of the blood, and only in small proportions in the serum; and by using the blood just as it comes from the animal, we seem to have everything to gain and nothing to lose. Furthermore, Hallion has preferred to prepare the blood with glycerin, instead of carbolic acid, since physiology and therapeutic experience have both shown this substance to be an excellent preservative of delicate biologic properties belonging both to the humors and tissues of the body.

Again, we know that in general terms the herbivoræ are preferable among the mammiferæ for serotherapic purposes: the sheep, or a horse, for instance, is better than the dog. It is also necessary not to begin using the humors of an animal until a month has elapsed since the removal of the thyroid gland.

Very satisfactory results can be had with comparatively small doses of this hematothyroidin preparation. Thus one teaspoonful,

once or twice a day before meals, in a little water, has been frequently used, although larger amounts have been given without any ill effects.

Enriquez prescribes it in the following way: Three teaspoonfuls a day, the first week; three dessertspoonfuls the second week; and tablespoonful doses the third week.

The results of the treatment are immediate and striking; the patient feels better in general, and the tachycardia and trembling improve, although the exophthalmos is slower to yield. On the other hand, some cases seem to resist the treatment; thus when there is goiter that has become complicated with the symptoms of Graves's disease, the treatment shows less evident results than in cases of pure exophthalmic goiter.

We are now in possession of a sufficient number of cases treated by this method and by different physicians, to be able to say that the results it gives are so evident that it is not possible not to admit that there is something specific in this form of serumtherapy of exophthalmic goiter.

SERUMTHERAPY OF HEMOPHILIA

In spite of a great deal of research, the true nature of this singular disorder called hemophilia remains a mystery. For this reason Weil has taken advantage of the presence in his wards during several months of a hemophilic patient, who lost blood for two weeks from the mouth after the extraction of three teeth, to go over the question afresh, and make some very important pathogenic and therapeutic experiments.

As is so often the case with hospital patients, very little could be ascertained about the hereditary antecedents of this man; it was therefore not possible to learn whether he belonged to the usual category of hereditary family hemophiliacs, for it is known that this disorder generally appears in certain families and their descendants. Thus with the Mampel family, which has been now followed medically for eighty years, there were, among 212 members, of whom 111 were males, 37 cases of hemophilia, all among the males, as is the rule.

A remarkable point in this connection is that a hemophilic man marrying a healthy woman, begets healthy children; whereas the women of hemophilic families, who do not manifest the disorder

themselves, pass it down to their male children. Still, women are not absolutely immune; but the disorder is infinitely more frequent in the male sex. When women have it, it shows a predilection for the genital sphere and appears during the incidents of genital life.

Three theories have been advanced to account for hemophilia: (1) An aqueous state of the blood; (2) disordered coagulation; (3) degenerated vessels, which do not contract.

The first is apparently wrong. Morphologically the blood is normal; there is intense anemia after hemorrhage, but the loss is quickly repaired. There is no leukocytosis. The percentage of water, osmotic pressure, and alkalinity are normal.

But the second theory is much more interesting, as it seems that hemorrhage with these patients is due to lack of power of coagulation. Fibrin is present in their blood in proper amounts.

Weil has ascertained, by the direct opening of veins in such cases (on which occasions, curiously enough, they do not bleed more than other persons) that with them the escape of blood is different from the normal; in the latter instance the blood comes through the cannula drop by drop, and soon slows up and stops, whereas in the former it flows quickly, and continues to flow as long as the cannula is left in place.

Such blood collected in a vessel takes a long time to coagulate, and does so in a manner of its own, quite distinct from that of blood in a normal condition.

Chloride of calcium quickens noticeably the coagulation of the blood of these patients; in Weil's case the flow from the mouth was checked by the administration of this drug during three days at doses of 90 grains (6 grams) per 24 hours, combined with local applications of adrenalin.

But the interesting and important side of Weil's paper lies in his experiments on hemophilic blood with the serum of the normal blood of men and of animals.

Thus he found that whereas hemophilic serum had no effect on the coagulation of normal human blood, the serum of the latter had a most pronounced effect on hemophilic blood; the addition of from 3 to 5 drops of normal serum to 3 c.c. of hemophilic blood, made it coagulate quickly and in all points as it rightly should.

He next ascertained that the same result, though somewhat less

evident, can be obtained *in vitro* with the serum of animal blood (steers, rabbits).

An important point to be mentioned here is that such normal serum has to be added to the hemophilic blood in very small amounts; as soon as this quantity exceeds a certain point the opposite effect is produced!

From these experiments he concludes that the lack of coagulation with these patients is not due to the presence in their blood of anti-coagulating substances, but to the absence or alteration of certain normal substances, which, when supplied in minute quantities restore normal coagulation to such blood.

Since, now, hemophilia is a family disorder, it appears that the absence or alteration of a secretion may constitute a functional deformity, and that together with anatomic deformities we must make room for a class of functional deformities. It is probably that in this way the transmission of this peculiarity by women and not by men can be explained.

Proceeding from these experiments Weil applied the results to his patient. He first studied the coagulation of his blood without treatment. Next he successively examined it after intravenous injections of 15 c.c. of steer serum, 10 c.c. of human serum, and 15 c.c. of animal serum again, at considerable intervals apart, the experiments extending over three months.

From these experiments he found that the results with his patient were what he had anticipated; his blood regained its coagulating power and resembled that of a normal person; the animal serum was less active than the human; the results of this species of vaccination, evident in a couple of days, did not extend beyond a month, by which time the blood had ceased to coagulate again.

Weil regrets that he has not yet had occasion to apply his discovery to a hemophilic patient in a period of hemorrhage; he has, however, treated his present patient to such effect that the man, who during his previous history had always bled for prolonged periods whenever his tissues were lacerated in the slightest degree, was able to have another tooth out, for the first time in his life, without any greater loss of blood than an ordinary person.

It is therefore probable that in this way any hemophilic patient having to undergo an operation, could be prepared to such purpose that he could stand it as well as any other patient.

Pediatrics

INFANTILE DERANGEMENTS DUE TO IMPROPER BREAST FEEDING, WITH SUGGESTIONS FOR THE PREVENTION AND TREATMENT OF CONSTITUTIONAL DISORDERS, SUCH AS RICKETS AND MARASMUS

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There are a great many points requiring emphasis in the management of a nursing infant fed with human milk. Many of these points are misunderstood, and in consequence, gastric and intestinal disorders frequently develop, and these if neglected may give rise to serious damage to the structure of the infant and in many instances may result fatally.

Two of the most frequent conditions met with in the children of young, inexperienced mothers, for which the physician is usually demanded, are underfeeding and overfeeding.

Underfeeding.—This condition is very commonly met with. An anxious and nervous mother who devotes her whole time to the baby usually develops anemia. The anemic pallor which is apparent is also transmitted to the milk and manifested by scanty milk secretion. Fat globules appear under the microscope (Fig. 1) to be irregular in size and scant in number, so that we see a thin emulsion. There is a very low percentage of fat and an excessive quantity of casein, noted by chemical examination.

When we study the chemical deficiency of the blood in an anemic woman we find that it is lacking in oxyhemoglobin; and this deficiency in the red blood cells plus the absence of iron is responsible for the weakened state of the woman. This condition is

brought about primarily by loss of sleep and by remaining indoors and leading a distinct sedentary life. A woman cannot eat and nourish her body properly if deprived of sleep, and it is this condition which renders her anemic and impoverished. What has just been described concerning the blood, applies equally well to the state of her milk. Such thin milk lacks vital elements, principally fat, and when fed to an infant results in tissue starvation, otherwise known as *underfeeding*. There are other causes which can induce this infantile starvation,—principally worry and anxiety due to the nervous system of the nursing mother. A woman subject to fainting spells or to epilepsy, or the common type of neurasthenic

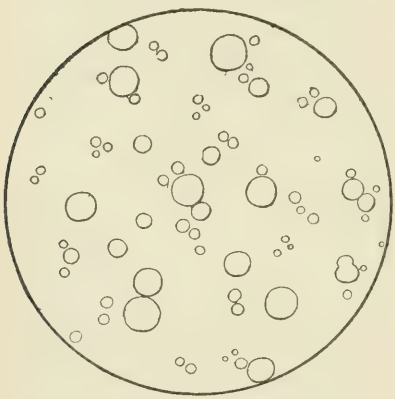

FIG. 1.—Microscopic appearances of a thin milk emulsion from an anemic woman. Her infant was not gaining weight.

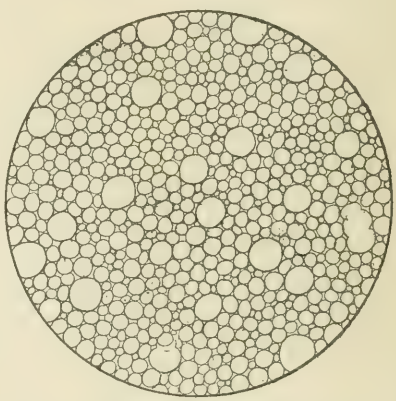

FIG. 2.—Microscopic appearances of a milk emulsion containing a high percentage of fat and causing gastric disturbances and vomiting in the infant. The infant was gaining in weight. The mother ate too much meat and took no exercise.

woman may be the cause of infantile starvation. Women with organic diseases, such as pulmonary tuberculosis, chronic Bright's disease, heart disease, and principally women with pelvic disease (who still bleed) and those who menstruate regularly, are the ones who usually secrete a poor milk; prolonged feeding of this may result in infantile atrophy and its allied disorders.

A starvation stool in an underfed infant is usually of a spinach-like consistency; it is green in color and not infrequently contains white particles of undigested curd, consisting principally of casein. The stools are thin and have very little consistency. At times, underfed infants have distinctly watery evacuations con-

taining mucus. Such infants do not gain in weight, and frequently lose weight; hence, a positive guide as to the development of an infant can be seen by watching the weight from week to week.

The surface temperature is usually cold. The circulation in the hands and feet is poor and the body temperature, taken in the rectum, is usually between 97° and 98° F. We may, therefore, say that a subnormal temperature goes with lowered vitality, induced by not receiving enough food.

Overfeeding.—This condition is also a very frequent cause of trouble. An infant will receive the breast and owing to some disorder such as colic, the mother believes the infant to be hungry. If this colic occurs one half or one hour after nursing, as is frequent, the infant often receives another meal to quiet it. In my children's service it is quite common for a mother to admit having nursed her infant every half hour for days and nights. Nor is it uncommon to have a mother report that the baby was restless and could not sleep without being nursed, and therefore the baby received the breast to quiet it. This simply means one continuous series of meals with their baneful results due to overfeeding. (Fig 2.)

We find that the majority of young mothers during the first few months of an infant's life, interpret every cry to mean only one thing—hunger. If they would stop to think that intestinal fermentation causes colic, or most often a disordered state of the bowel induced by constipation causes pain, the mistake of too frequent feeding would not be made. Pain, hunger, thirst, sleep, the burn of an excoriated skin, are conditions all reported by the infant by crying. Hence it is apparent that the cry being an infant's expression of ailment, such as pain, also its expression for hunger, we must exclude all else before deciding that the baby is hungry. This can easily be done if regularity in feeding and a proper interval has been given.

Symptoms of Underfeeding.—An underfed infant is one that will cry and appear fretful by day and sleep very little at night. Such infants usually have their fingers in their mouth and suck on them. They drink everything, even water, very greedily. As a result of gulping their food very fast, they are subject to colic, and not infrequently they vomit.

Symptoms of Overfeeding.—Frequent vomiting of sour, undigested curdled milk, with associated dyspeptic colic, is most frequently noted. Such infants usually suffer pain in the colon and, as previously stated, are fed by the mother or wet nurse instead of receiving an enema to relieve the distended gut of its fermenting contents.

The temperature as a rule is elevated and, owing to stagnant residue, dyspeptic fermentation may rise with associated temperatures of 102° to 105° F. Thus we can see that in the one instance while in the underfed child a subnormal temperature prevails, the overfed child will have pyrexia due to gastric and intestinal fermentation.

The stools of an overfed child are usually yellowish and contain less particles of visible casein than those of the underfed child. As a rule, there is mucus of a glairy character. The stool also contains a great deal of gas and when passed the evacuations are of an explosive character.

The stomach of an overfed child is usually distended and tympanitic on percussion. As a rule the legs are flexed on the abdomen, the infant is uncomfortable and hence cries considerably. Emaciation is not present, and the infant may even show a slight gain in weight. The circulation, as a rule, is quite good. The hands and feet are usually warm.

Prognosis.—The outcome of a case of underfeeding depends on how much can be done to produce healthy milk. This can be accomplished by combining the strictest attention to hygiene, by changing the mode of living—which means frequently to separate the mother from the child except at feeding time. If such conditions cannot be enforced, or when prejudice or ignorance prevents the intelligent management of the case, it is our duty to warn the parents and especially the mother of the danger that may come. I have seen the severest types of rickets, involving the bones of the head with rickety ribs and rhachitic extremities, spinal curvatures, bow legs, and decayed teeth, all decided evidences of improper nutrition, and due frequently to defective human lactation. Atrophic conditions, such as are commonly termed marasmus, frequently result in a dyspeptic infant, the foundation of which can be traced to abnormal feeding during the first year of life.

Chronic dyspepsia is frequently brought about by overfeeding during infancy. Stuffing the infant to make it fat and thus overfeeding it and overtaxing the weakened digestive tract is responsible in many instances for a chronic dyspepsia. Atony of the stomach and bowel is frequently traceable to faulty infantile nursing. Enlargement of the stomach (acute and chronic dilatation) is frequently brought about by over-distension, due to overfeeding and consequent fermentative disorders. The large majority of cases of so-called "second summer" worriments are simple dyspeptic conditions due to careless infantile management. It is therefore evident that the management of a breast-fed infant is not without danger, especially amongst the inexperienced and those who are not familiar with the outcome and the dangers subsequent to the overfeeding and underfeeding. It is surprising to see the change in an infant when strict regularity is enforced. In some instances the life of the child may depend on the rapid withdrawal of the mother's breast and the substitution of a wet nurse, if breast milk is demanded; in most cases artificial feeding will be demanded.

If by chemical examination we find that the breast milk continues low in fat and high in proteids, in spite of the change in the mode of living prescribed for the mother, or if the infant's condition does not respond and improve within several weeks after the hygienic conditions of the mother and child are improved, we must consider a radical change in the infant's food, such as total weaning and the substitution of artificial food.

Treatment.—A regular feeding interval should be ordered. I insist on a two-hour interval in a very young infant. For an infant three months old a feeding interval of three hours and no less should be prescribed.

Ample regard for the night's rest should compel us to omit at least two feedings during the night. We must not cater to the whims of a young infant and nurse it simply because it cries. We must consider the amount of milk that the infantile stomach can assimilate. If an infant cries soon after nursing and shows symptoms of hunger as described above under underfeeding, a radical change in the mode of living of the mother is indicated. Such change consists in active outdoor exercise, so that the mother receives oxygen. The old-time story that the mother works con-

tinually in the house, and is on her feet is no proof that she is living according to proper hygienic means. The stimulating effect of oxygen plus active exercise will frequently produce rapid improvement. Such improvement can be noted as transforming a soft, flabby mammary gland with scanty milk secretion into one having firm glandular development from which a good flow of milk can be expressed. In addition to an active outdoor life in which we aim to give the mother oxygen for its immediate effect upon her blood and also upon her milk, we also give her a certain amount of mental rest. That is, in isolating her for a short time from the mental care of her baby we remove the tension to which the nervous system has been subjected. It is this tension, commonly called strain due to worry, which causes so many cases of physical collapse. This collapse due to exhaustion following loss of sleep, loss of food, and constant strain, is called by neurologists either neurasthenia, hysteria, or nervous prostration. If rational means are insisted upon, in which exercise out of doors, rest at night, and good wholesome food for the mother is provided, then the milk supply will be more normal than when these important points are neglected. Deficient or scanty milk can be easily remedied. It is but necessary to order in addition to the above-named hygienic measures, a drink of hot milk, soup, broth, or tea with milk several minutes before each nursing. Hot oatmeal gruel (strained) will frequently stimulate the milk supply.

If the chemical examination shows deficient proteids, this can be remedied by ordering the mother to eat meat and eggs, in addition to cereals. If the fat in her milk is deficient, the addition of cream, and butter, and rich milk, and the yolks of several raw eggs daily will increase the percentage of fat.

If the proteids are too high, as evidenced by the high percentage of casein, active exercise will reduce them. A sedentery life usually increases the proteids. If the total quantity of milk is insufficient, give large quantities of liquids, especially before each nursing.

Mixed Feeding.—In some cases when the breasts fill very slowly, the question will arise as to whether or not maternal feeding should be continued. In such cases wherein the anemic state of the mother and the scanty milk supply are not amenable to treatment, the following suggestions are given: Utilize as much breast milk as

FIG. 3.—Rickets in a breast-fed infant.

possible, and substitute a bottle instead of every second or third nursing. Thus we give the mother more rest and still utilize her scanty milk supply for alternate feedings.

Treatment for the Overfed.—The strictest attention to the interval between feeding is imperative. In addition thereto smaller meals should be prescribed, so that the infant receives the breast less frequently and less in quantity. An average healthy infant will nurse between five and fifteen minutes. In an overfed infant with dyspeptic symptoms certain points must be exercised: first—insist upon slow nursing. This should be accomplished by withdrawing the breast from the infant's mouth several times during one meal. The meal should extend over twelve to fifteen minutes, no less, rather somewhat longer. We must not permit an infant to gulp its food hastily. It is this too rapid feeding that causes dyspeptic disorders in most cases. In some instances it is wise to order sweetened rice water or barley water after the nursing act is completed. Some infants do not assimilate starch, be it ever so diluted. In fact, the slightest quantity of starch will frequently give rise to vomiting or fermentative dyspepsia. In such instances we can give sweetened water, about one ounce after the infant has nursed, instead of rice water.

Concerning Rickets.—It must not be supposed that because an infant receives breast milk that all will go well. Rickets will frequently result when a milk deficient in proteids and fat is given for a long time. In my book on infant feeding, third edition, page 282,[1] I illustrate a case of rickets with beaded ribs in a breast-fed infant (Fig. 3). This infant had marked kyphosis and was one of the worst types of rickets encountered in my experience—an infant brought up on breast milk, in a poor family, living in a tenement house with poor hygienic surroundings, which no doubt devitalized both mother and child. This is very evident when we associate the progress and development of this child in contrast with one brought up in good hygienic surroundings. What has been said concerning rickets applies equally forcibly to the prevention of marasmus.

[1] Published by F. A. Davis Co., Philadelphia. The accompanying illustration is reproduced through the courtesy of the F. A. Davis Company.

Marasmus.—I have frequently seen infants continuously fed with abnormal breast milk in which the underfeeding resulted in marasmus. In some cases the prejudice against weaning was so marked that mothers continued to nurse in spite of warning to the contrary. Atrophic conditions, with coincident loose, watery, and curded stool, are frequently noted in conjunction with prolonged lactation.

Prolonged Lactation.—In some instances mothers will persist in nursing their infants more than a year. A case of this kind came to my notice in which a mother nursed her infant twenty-eight months, and as a result the infant was backward in development. The excuse offered by some mothers for this prolonged lactation is that it will prevent conception during their nursing period.

A wasted and enfeebled body results from insufficient food. This applies equally as well to a thin, insufficient breast-feeding. When the proportion of nutritive ingredients, such as the fat and proteid, are reduced to a minimum, instead of supplying energy and heat, as fat should, we have a cold and a subnormal bodily condition. When the proteid is abnormally low, it is evident in such symptoms as delayed dentition. The bones of the spine, the bones of the extremities, and the bones of the skull are thin, pliable, yield on pressure, and give such symptoms as craniotabes. Or they may be thick and spongy, as evidenced by the enlargement of the epiphysis of the bones in the arms and legs. This abnormal ossification is the result of deficient breast milk. A fluoroscopic examination made with the x-ray apparatus will show that the bones are not normal. What can be seen and felt in the osseous system is even more apparent when the muscular structures are examined. The muscles have a soft doughy feel and show a lack of tone. There is a general flabbiness. The muscles hang loosely and are easily indented on pressure. The skin and fatty layer seem to be very thin. There is no firm cellular tissue palpable. There is usually a tendency to local skin infections, so, for example, such children have usually one or more furuncles on the scalp.

Distended Abdomen.—In some cases instead of a retracted abdomen there is a fulness and marked distension due to intestinal fermentation. When such muscular distension remains it can

result in a permanently enlarged pendulous belly. The temperature is frequently as low as 100° F, but it may reach 105° F. Whenever a specimen of urine can be obtained it is important to examine it for the presence of indican. I have come to lay as much stress upon a positive indican reaction as showing the presence of putrefaction and stagnant fecal matter in the intestine, as I usually rely upon the presence of the diazo reaction when suspecting typhoid in a given case.

Neurology

HYSTERICAL NEUROSES OF THE STOMACH, WITH THE REPORT OF A CASE OF RHYTHMICAL BORBORYGMI OF HYSTERICAL ORIGIN[1]

BY JAMES HENDRIE LLOYD, M.D.

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I am fully aware that hysterical affections of the stomach are not likely to be popular at the present day. So far as I have followed the current of contemporary medical thought, affections of the stomach are rapidly passing out of the domain of clinical medicine into the control of the surgeon. It seems even probable that there will soon cease to be any very wide field for the mere physician in gastric pathology, unless it be to make a hurried and half-hearted diagnosis and immediately to call in the surgeon. If the medicine-man is in doubt about the diagnosis, all the better; the surgeon stands ready to relieve him of all responsibility and to make an exploratory incision before he has barely had time to complete his percussions and his palpations.

The neurologists, perhaps, are not yet fully prepared to follow this surgical lead. We are still spunky about some of our clinical rights, and not ready to resign them. The day has not yet dawned when a neurosis can be cut out, although there are some ominous mutterings from the surgical camp; and only recently a prominent Philadelphia surgeon has made the direful announcement that hysterical affections too have probably an organic basis; and he seemed to imply that for his part he was ready to employ a knife.

Whatever may be our ultimate fate, and the fate of our patients, the fact remains that we may still exercise our prerogative of making

[1] Read at a meeting of the Section on General Medicine of the College of Physicians of Philadelphia.

a diagnosis; and it is in this humble spirit that I appear before you to-night.

The list of hysterical neuroses of the stomach (in which may be included also the abdomen) is a long one, and it admits here of but a brief review.

The condition known as *anorexie hystérique* was described in a classical paper by Lasègue[1] and, after him, under the title of *anorexia nervosa,* by Sir William Gull.[2] It is a condition which is not unfamiliar to neurologists, for it is often seen in various degrees in cases both of hysteria and neurasthenia. The patient declines to eat a sufficient quantity of food; there is absolute indifference, and this is the cause of wasting, which in some cases becomes extreme. With this malassimilation, the nerve-tone is absolutely demoralized, and this rejection of food becomes the critical element in the case. These cases may present the appearance of organic disease, especially if the anorexia is accompanied, as is sometimes the case, with hypersensitiveness about the epigastric region, a true hysterical gastralgia. I do not intend to dwell upon this condition, for, although an important one, it is too fully recognized to need description. We have all seen these wretched broken-down women, some of them weighing as little as eighty or ninety pounds, and resisting the administration of food as though it were a poison. The diagnosis requires caution: sometimes there is suggested a gastric ulcer, a carcinoma, a latent tuberculosis, or a Bright's disease; and in fact all these grave organic affections have been overlooked in such cases, and a serious mistake committed.

In passing, however, I wish to call special attention to the possibility of a concealed drug habit as a cause of such conditions. I have recently had under my care a lady in this state of neurasthenia with anorexia and wasting, who had been secretly taking an opiate which she claimed (I know not how truthfully) had originally been prescribed for her by a gynecologist. These drug habitués are only too apt to accuse some physician of being the cause of their downfall. Their accusations must be received with strict incredulity. But there is no doubt that a secret drug habit is often the underlying cause of anorexia, emaciation, and hysterical perversions. These

[1] De l'Anorexie Hystérique, Arch. Gén. de Méd., Apr., 1873, p. 385.

[2] Anorexia Nervosa, Trans. Clin. Soc., Lond., 1874, vol. vii, p. 22.

patients have phantom pains about the stomach, back, head, and limbs, which plague the life out of them, and out of their physicians, and prompt the constant cry for more drugs. The mental and moral state in some of these patients is far the most important one, and the physician who overlooks this element is in error. All these drug-takers are not moral perverts, as is often claimed. One of the gravest elements in their cases is the sense of remorse, of self-reproach, of helplessness in the grip of a tyrannous habit. They also become hypochondriacs, and may locate an imaginary trouble in the uterus, the heart, or the stomach, and present a most perplexing problem for diagnosis. The woman who takes opium, and comes gradually to have recurring paroxysms of pain about the pylorus and gall-bladder, may fool even the most elect diagnosticians.

Closely allied to anorexia and sometimes forming a part of the same clinical picture, is hysterical vomiting. In some patients almost everything that is eaten is vomited; in fact the vomiting becomes almost automatic and is repeated at regular intervals. It persists even when the stomach is empty, and it is not accompanied with the feeling of nausea. In the absence of food in the stomach to be vomited the patient will continue to retch, and go through the motions until only a little tenacious mucus is dislodged from the depths of the throat, as was noted by Naudeau [3] as long ago as 1789. These patients, as would seem inevitable, are often extremely emaciated, although some authors report just the reverse. There seems indeed to be no uniform rule about this. In some hysterics, in spite of constant vomiting, or, as it may be called, pseudo-vomiting, the nutrition remains good. But in the best marked case of the kind that I have ever seen, the wasting was extreme. This was in a young woman whose condition dated from the day when she had taken by mistake a dose of nitrate of potassium (ordinary saltpeter) in mistake for Epsom salt. She and her family had been greatly alarmed, supposing that the dose would prove fatal, and the vomiting which began at the time continued as a daily, and even hourly, occurrence for almost one year. This young woman's emaciation was so great that it was feared she would die, and in

[3] Sur une Maladie nerveuse, Jour. de Med., Chir., Pharm., etc., Juillet, 1789, p. 197.

fact fatal cases, I believe, have been reported. The affection was promptly cured by removal from home, isolation, rest-treatment and suggestion.[4]

So automatic and mechanical is this act of vomiting that it seems to demand a special name, for the act is not always a true act of vomiting. In some patients the introduction of food even into the esophagus excites its rejection, and the bolus of food, or even of fluid, does not pass into the stomach at all. It gets no farther than the esophagus, and the condition has not unaptly been called *esophagismus*. There is an underlying state of hypersensitiveness of the gullet, the whole complex act of rejection being dominated by a mental state. It is a psychosis. The resemblance of this condition to some organic disease of the esophagus or of the cardia is very striking. In the case already referred to, the patient, while lying on her side, had constantly under her face and chin a large linen or muslin cloth. As she lay thus, she had at frequent intervals a violent retching, which, as her stomach was almost always empty, brought up only a little mucus and saliva. After taking food, which she did in very small quantities at a time, she almost immediately rejected it by a sort of regurgitation, rather than vomiting, so that probably all the food did not really enter the stomach. Some food, of course, she retained, but her relations said that the gagging and retching were continued at intervals of a few moments all during her waking hours. They were worse when the patient was observed by strangers. They were not attended with nausea. The patient's appearance was sufficient proof that her stomach received and digested the smallest amount of food necessary for life. She was extremely emaciated; the muscles of her limbs were wasted almost as in muscular atrophy, and the seat of a violent tremor, and the belly was scaphoid.

One of the most curious of all gastric neuroses is the habit known as *merycism,* or chewing the cud. This *ruminatio humana,* or as the Germans call it, *wiederkauen bei menschen,* is closely allied to the condition just mentioned, *i.e.,* esophagismus. It is a neurosis, associated sometimes with various forms of mental and nervous disease. Thus the late Dr. Kerlin of the Elwyn Institute

[4] Hysterical Tremor and Hysterical Anorexia (Anorexia Nervosa) of a Severe Type. Am. Jour. Med. Sci., Sep., 1893.

for Feeble-Minded Children told me that he had seen it in an idiotic boy. Chatelet[5] made a similar note; and Rossier[6] reported an instance in an epileptic. I believe that most if not all cases, if critically examined, would be found to occur in neurotic patients. In the past the subject has appealed rather too much to the wonder-loving element in our profession, and these cases have not always been carefully studied and criticized. An exception should be made in favor of an admirable report of two cases by Dr. David Reisman before this College. The subject, curiously enough, is not a new one. According to Gratziana,[7] who has written a recent paper in Italian, it was first described in 1618 by Fabricius de Aguapendente. Ludovici wrote in 1678 *De Ruminatione Humana Brutorumque,* showing how the man and the brute were associates in this matter in the minds of our predecessors. Peyer (he who described the patches) is credited with twelve observations in 1685; and he supposed there was a dilatation of the esophagus constituting an *antrum cardiacum,* or ante-stomach. Thus the attempt was made to give these poor hysterics an extra stomach, like a cow, in order to explain a mere mental perversion; but even this may be better than cutting out this viscus in order to cure these patients. The change from the seventeenth to the twentieth century is not altogether in our favor. Peyer even seemed to have an inkling that there was a psychical element in these cases; and Schmid, who wrote an inaugural dissertation in 1709 on the *aegrum ruminantem,* recognized a hypochondriacal taint. A case was reported to the Royal Society nearly 200 years ago. In our own time the eminent physiologist Brown-Sequard is said to have formed this habit on himself as the result of an experiment. Not a few cases are now on record in medical literature, as reference to the Index Catalogue will readily show. But perhaps the most bizarre paper was that of the late William A. Hammond[8] who reported that he had trephined a weak-minded young man for merycism. He first took out a button of bone on one side of the skull, and then on the other side, to make sure, I suppose, that he had hit the ruminating center

[5] Mem. et Comp-rend. Soc. de la Méd. de Lyon, 1863, ii, 66.
[6] Jour. de Conn. Méd. Prat., Paris, 1862, xxix, 181-184.
[7] Riforma Med., 1898, xiv, pt. 3, 436, 446.
[8] Jour. Nerv. Ment. Dis., 1894, 21, p. 680. Abstract.

in the brain cortex. The operation cured the patient—a brilliant example of brain-surgery!

In merycism the act consists in a regurgitation of food into the mouth, when in some cases it is again chewed and once more swallowed. There is said to be some gustatory satisfaction; the patient enjoys the act; he has two square meals instead of one, and in confirmed cases there is a constant disagreeable odor of the acid stomach-contents about the mouth and person of the merycole. He smells like a puking baby.

Many theories have been invented to explain this condition; such as a reverse peristalsis, or an "irritability of the vagus nerve." But the real explanation is probably found in the theory that the practice is simply a bad neurotic acquired habit. Several cases have followed one or other of the infectious diseases, as typhoid fever, or measles.

The point which I wish to emphasize in regard to these various neuroses is that they are closely allied to habit spasms or *tics*. They probably begin as a voluntary act, or at least under the influence of some mental state, and they come in time to be in a sense automatic. That they are not entirely outside of the domain of volition is proved by the fact that they are often cured by mental impression. They seem to center about the cardia; and this is the gastric region which is most under the control of volition and most within the domain of consciousness.

Briquet, in his work on hysteria, has described a very rare condition which he calls *paralysis of the diaphragm;* and Duchenne of Boulogne has described a *contracture of the diaphragm;* it is the cause of embarrassed breathing and asphyxia, and is doubtless allied with the peculiar rapid respiration seen in some hysterics. This motor mechanism of breathing may thus come under the dominion of an hysterical perversion, just as the motor apparatus of the stomach does.

In this connection we must not ignore those curious *phantom tumors* which are occasionally seen in hysterical patients. Gilles de la Tourette,[9] in his work on hysteria, has a long discussion of this subject. The belly suddenly balloons up with an enormous tym-

[9] Traité de l'Hystérie, 2d part, ii, chap. 17, p. 340, et seq.

panites; this is true gas, although some observers have been inclined to doubt it. But Sir Benjamin Brodie proved it by inserting a tube into the rectum and drawing the gas off—certainly a convincing demonstration. When associated with pain we have the state which the English have called *spurious peritonitis.* It is well known that such a condition has been mistaken for pregnancy—the so-called *false pregnancy,* which may even deceive the patient herself, as in a case which I saw some years ago in which the woman prepared for her lying-in and went to supposed full term with nothing but air in her abdomen. Such a condition has indubitably some connection with the mind as a cause, although it is difficult to explain it. I once saw an old lady who just a few hours before her husband's funeral, when she was, of course, under deep emotion, was taken with a sudden and alarming distension of the abdomen; the belly was greatly swollen and painful and there was great oppression of breathing. It passed off in a short time.

This brings me at last to the case of my patient whom I exhibit here to-night.

This case presents an example of a hysteroneurosis of the stomach or abdomen of a quite nondescript character. I have called the condition one of *rhythmical borborygmi,* rather for want of a better name than because I think it is entirely appropriate. The abdomen is the seat of a rhythmical movement, made by the diaphragm and abdominal muscles, and associated with a loud to-and-fro sound which is located in the epigastric region. This movement, if attentively studied, is seen to be synchronous with the act of respiration. It is caused by deep heaving movements of the diaphragm: as the diaphragm moves up and down, the abdominal muscles contract violently, and thus there is an exaggerated motion which is confined largely to the abdomen. The breathing is not affected; there is no forced respiration, no dyspnea nor anxiety. The condition is one primarily of forced and rhythmical movements of the diaphragm, and may thus be allied to the contracture of the diaphragm observed, as already said, by Duchenne of Boulogne in an hysterical subject. The affection is thus seen to be located rather in the abdomen than in the thorax, and the loud bruit is evidently caused by the movement of air in one or other of the abdominal organs.

This movement is almost constant, especially when the patient is up and about, or even sitting up. It disappears completely when the patient is lying down, at which time the respirations are even and without effort and perfectly normal, and the sound ceases. It reappears promptly, however, even while the patient is lying down, if she is observed and her case studied.

The mechanism of the sound is obscure. It is evidently made by air passing to and fro through a narrow structure; but whether this is the pylorus or whether the seat of the sound is in the transverse colon is obscure.

The case nearest resembling it that I have found in the literature is one reported by Lécorché, which he called an example of "rhythmical borborygmi" in an hysteric, and from him I have borrowed that term.[10] In Lécorché's patient the sounds were rhythmical, and consisted in a sort of bruit, of a to-and-fro kind, first of a scraping sound, then of gurgling, and succeeding regularly from right to left and from left to right. This description tallies fairly well with my case.

The clinical history, written by the resident physician, Dr. Powell, is as follows:

D. A., female, aged 23 years, unmarried. Admitted to the Methodist Episcopal Hospital, January 20, 1906.

Her father and father's family are of nervous temperament. Her father has had paralysis agitans and has been in poor mental condition for 8 years. Father's sisters died of consumption. Mother, two brothers, one sister (twin), living and well.

The patient was born in Philadelphia. She went to school until her fourteenth year; then to work; first in a shirt factory, later in a cigar factory as a stripper. She remained there 3 years; then had what was diagnosed as "walking typhoid." On returning to her work she was again taken ill, and states that she had what the doctor called a "nicotin ball" in her stomach. She felt as though there was a lump in her stomach. The region over the epigastrium was so tender that she could not endure the weight of her dress. She was sent to bed for 13 weeks, and finally passed by bowel the so-called "nicotin ball" which she asserts looked like a bunch of worms.

[10] Etudes Médicales, 1880, p. 540, quoted by Gilles de la Tourette, de l'Hystérie, 2d part, ii, p. 341, 342.

She passed some mucus at the same time. She also states that she had an attack of grip during her 13 weeks in bed. This all occurred 5 years ago. Soon after getting about the patient states that she had a peculiar sensation in lower abdomen—a heavy pressing-down feeling. She began to have distress in her stomach about this time, and underwent a slight operation, probably a dilatation and curettement for the womb trouble.

She did not vomit, had no pain after eating, no belching of gas or acid eructations, but merely an uncomfortable feeling which has continued more or less constantly up to the present time. She has done no work since that time.

Menses came on at the age of 14 years; were painful and lasted 3 to 4 days. They were very irregular, and there has been as long as 6 months between periods. Last menstruation November 17, 1905.

The patient states she has had some rheumatism and some cardiac trouble. History very indefinite. No history of vertiginous or syncopal attacks. This story, it will be observed, has some very bizarre features.

The patient first noticed a peculiar noise in her stomach about 6 months ago. At first the noise was slight and did not annoy her; but it became more and more marked from week to week, though it would disappear at times for a few hours when she remained very quite. It was always worse on exertion and especially when the patient was up and dressed. She kept about until Christmas time when she took cold and the condition became more marked, so that she could not go out on the street or appear among friends.

On admission the diagnosis of the family physician was atonic dyspepsia and pyloric incompetency.

Condition on admission: A peculiar rhythmical churing motion of the stomach is seen on assuming other than the horizontal position, accompanied by a swashing sound as of fluid and air in active motion within the stomach itself; the point of greatest intensity seems to be at the pyloric orifice. When the stomach is quiescent its lower border is ½ inch above the umbilicus, but when distended or when in motion it reaches a point 1 inch below the umbilicus (Fig. 1). The entire organ apparently takes part in the descent. That is, there is rather a transitory gastroptosis than a distinct increase in size.

The patient does not complain of indigestion; suffers no pain; never vomits after eating, and while quiet in bed the stomach remains quiet. But on sitting up in bed or standing on her feet, she presents this peculiar borborygmus above mentioned.

Physical Examination.—The patient is fairly well nourished and without external marks of noteworthy interest.

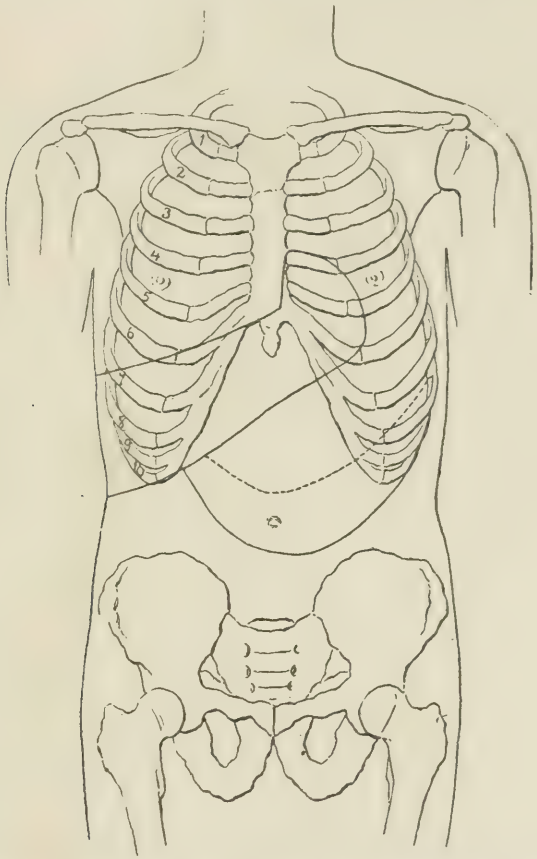

FIG. 1.—The dotted line indicates the lower border of the stomach when quiescent; the unbroken line, the lower border when in motion.

Heart: Outline, third rib, left border sternum, middle clavicular line. Apex beat, fifth interspace inside middle clavicular line. No murmur.

Lungs: Negative.

Liver: Sixth rib, costal margin, and negative.

Spleen: Not palpable.

Kidneys: Negative.

Abdomen: Flaccid; no pain, tenderness or rigidity except for voluntary act.

Reflexes: Normal.

Nerves: There are no sensory disturbances, nervous stigmata or hysterical tendencies demonstrable except those associated with the stomach. There is no history of convulsive attacks or exhibition of hysterical tendencies.

Urine: Specific gravity 1024, no albumin, sugar, or casts.

Blood: Erythrocytes, 5,420,000; leukocytes, 6,780; hemoglobin, 80 per cent.

Gastric analysis: Total acidity, 30 c.c.; free Hcl., 0.3; lactic acid, negative; microscopic examination, negative.

In the very helpful discussion of this case before the Medical Section of the College of Physicians of Philadelphia, various suggestions were offered to account for the mechanism of this bruit; as, for instance, stenosis of the pylorus, hour-glass contraction of the stomach, etc. It was pointed out, however, that there was nothing in the patient's history to indicate organic disease of the stomach, such as an ulcer or inflammation, which might by producing contracting scar-tissue lead to distortions and deformities of the organ. But even supposing such organic lesion to exist, the difficulty still remained of accounting for the peculiar bruit. It is, of course, possible to conceive of an hysterical symptom engrafted, as it were, upon an organic one, a thing not unusual in neurological practice. Thus if organic lesions, such as contractions about the pylorus, exist, the peculiar movement of the diaphragm might nevertheless be a neurosis.

The general consensus of opinion, however, was in favor of the whole condition being one of hysteria.

This patient entirely recovered after a few weeks' treatment in the hospital. The treatment was almost entirely by suggestion, with the use of a tonic mixture merely by way of a placebo.

Surgery

TUMORS OF THE MAMMARY GLAND, WITH SPECIAL REFERENCE TO THEIR DIAGNOSIS, PROGNOSIS, AND TREATMENT

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ANATOMY OF THE MAMMARY GLAND

As a knowledge of the normal structure and relations of the breast is essential for an adequate conception of the mode of development, extent, and effects of the neoplasms which affect it, as well as for a thorough understanding of the principles upon which their rational surgical treatment depends, it is advisable to review briefly its most important anatomic characteristics.

Until recently it has been customary to describe the breast as a hemispherical body extending from the second or third rib above to the sixth or seventh rib below, and laterally from the outer side of the sternum to near the anterior border of the axilla. It is now known, however, that this description by no means accurately defines the limits of the organ, for it has been shown that prolongations of glandular substances may extend upward toward the clavicle, downward toward the external oblique muscle, outward into the axilla, and inward to the sternum. Although the greater part of, and indeed not uncommonly, the entire gland, lies in the superficial fascia, being fastened to the deep fascia or sheath of the pectoralis major by loose areola tissue, processes of glandular substance sometimes extend into this deep fascia, or even through it into the muscle itself.

A well-developed mammary gland consists of fifteen to twenty

lobes, supported by fibrous tissue and embedded in fat. The anterior surface of this glandular mass is rough and uneven, presenting elevations from the apices of which bands of fibrous tissue descend into the interior of the gland between its lobes and also extend outward to the skin. Each lobe has its own excretory duct which, as it approaches the nipple, becomes dilated into a small sac, called the lactiferous sinus. As the sinuses advance toward the periphery of the nipple they become narrowed again, and perforate its tip by means of minute openings about 0.5 mm. in width. The acini—of which these lobes consist—are lined with cubical epithelium, as are also the minute ducts; the main excretory ducts, however, are lined with columnar cells.

The mammary gland is drained by a complex lymphatic system, much more intricate and extensive than it was formerly thought to be. There are at least six important and fairly constant routes; of these, three are superficial and three deep. In addition there are inconstant or accessory channels.

Superficial Lymphatics.—(1) The principal set of Sappey, already described, which forms a plexus at the areola, thence passing by main channels to the axillary glands.

(2) This set drains the skin over the sternal half of the gland, passing through the second and fourth intercostal spaces to discharge their contents into the nodes of the anterior mediastinum. Some of them occasionally interlace with those of the opposite breast, which explains the involvement of the axillary glands on the right side in tumors of the left breast, and vice versa (Reiffel, Oelsner, etc.).

(3) A cutaneous set of vessels, recently described by Poirier and Cunéo, which drains the upper part of the breast and passes directly over the clavicle, emptying into the supraclavicular glands.

Deep Lymphatics.—(4) This set begins in the mucous membrane of the milk ducts and acini, drains the deeper portions of the axillary half of the gland, joins the superficial set in the axilla, and forms with it a network which surrounds the axillary vein almost or quite up to the clavicle.

(5) A second deep set drains the deeper portions of the sternal half of the gland, perforates the second and fourth intercostal spaces, following the course of the internal mammary artery,

to empty, together with the superficial set, into the mediastinal glands. On the right side they intermingle with the lymphatics of the liver. Hence it is easy to explain the implication of the mediastinal glands, resulting in the prominence of the bone, or "sternal symptom" of Snow in cancers originating in the inner half of the gland.

(6) A third set drains the middle of the base of the gland and retromammary tissues. Some are lost in the fascia covering the pectoralis major, while others perforate the intercostal muscle to follow the intercostal vessels to the spine, thereby affording a ready explanation of those cases ultimately complicated with spinal symptoms, even paraplegia; such cases are wrongly spoken of as general infection, as they are caused by local extension along the lymphatics.

In addition to these usual avenues of infection in early or average cases of breast cancer, there are additional or accessory channels in cases in which the breast is immobile and adherent to the pectoral muscles beneath. Grossmann injected three times in 30 subjects a lymph-bearing vessel from the mammary gland which perforated the great pectoral, running between it and the pectoralis minor to empty finally into the subclavian or topmost axillary glands. Along the course of this vessel are several small retropectoral glands which become enlarged in 50 per cent. of all cases of cancer (Rotter).

Lymphatic Glands of the Axilla.—There are usually from 12 to 36 axillary glands, which are of the greatest importance from a surgical standpoint, as they receive the afferent lymph vessels from the arm, skin of the chest, mammary gland, and subjacent muscles. They are arbitrarily subdivided into groups:

(1) The humeral chain which follows and surrounds the axillary vein.

(2) The thoracic chain, which lies upon the inner wall of the axilla, being hidden, partly or entirely, by the insertion of the pectoralis major muscle.

(3) The scapular group, which follows the subscapular artery, and is found on the posterior axillary wall.

(4) The central group between the preceding chains.

(5) The subclavian chain, perhaps the most important of all, consisting of three to twelve glands situated above the tendon of the

lesser pectoral in the space of Mohrenheim, and receiving the afferent vessels from all of the axillary glands below. These glands are intimately associated with the axillary vein, lying to its inner aspect at the point where it receives the cephalic vein. The vein may be injured when removing the glands if enlarged, unless the removal is done under the eye by careful dissection.

In regard to the blood-supply and nerve-supply of the mammary gland, it will be remembered that numerous branches are given off to it from the internal mammary artery, and that it also receives branches from the long thoracic and occasionally from the acromiothoracic, both of which are given off from the axillary. The veins correspond to the arteries. The nerves come from the supraclavicular branches of the cervical plexus and from the fourth, fifth and sixth intercostals.

In the male the breast is very rudimentary, varying very much in size and symmetry. In the majority of cases it is placed over the fourth intercostal space, a little internal to the border of the pectoralis major. It is subject to inflammation and to the neoplasms which arise in the female breast, although much less frequently attacked by the latter affections. In fact, benign growths are most rare in the male organ. I never saw one. Cancer occurs not infrequently: I have encountered at least 4 typical cases of schirrus carcinoma. Recently I saw a growth of six months' duration, following traumatism, in a lad 16 years of age, with all the appearance and clinical history of sarcoma. Although its removal was advised and insisted upon the parents are still hesitating.

BENIGN TUMORS AND CYSTS OF THE MAMMARY GLAND

Much confusion has existed in regard to the classification of benign growths of the breast, and this is due largely to the fact that surgeons failed to classify them upon a strict histologic basis. Especially annoying has been the multiplicity of terms applied to those benign growths the chief constituent of which is fibrous tissue, but which also contain epithelium in variable amount. Thus Virchow, Monod, Gross, Cornil, and Ranvier, applied the name fibroma to these tumors, quite ignoring in their nomenclature at least, the presence of epithelial elements,—whereas Billroth, Green and Duplay used the term adenofibroma, and Broca called them adenoma

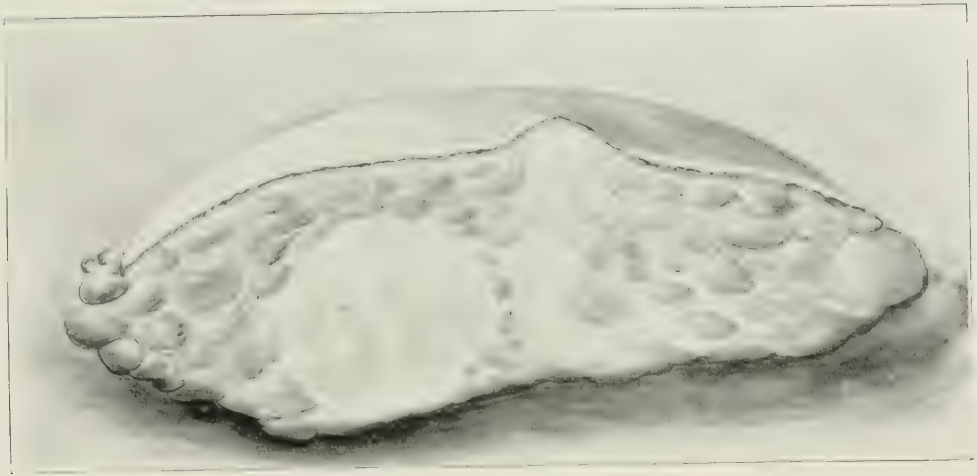

FIG. 1.—Periductal fibroma. Gross appearances. (Warren.)

(Figs. 1 to 9 inclusive have been reproduced through the courtesy of Dr. J. Collins Warren, of Boston, and the editor of the Journal of the American Medical Association.)

FIG. 2.—Periductal fibroma. Microscopic appearances. (Warren.)

with predominance of stroma. Adenocele is another name which has been given to them, particularly by the English. All of these latter appellations were designed to recognize the existence of epithelium in the tumor. Ribbert has recently included them, together with some other growths containing the same as well as different structural elements, in one group to which he applied the term fibro-epithelial. Warren, accepting this term of Ribbert's has divided the group into two principal types, the fibrous and the epithelial, according as one or the other type of tissue predominates. The fibrous type is still further subdivided—and upon a strictly structural basis—into fibroma, myxoma, and sarcoma, and the epithelial type into fibro-cyst-adenoma and papillary cyst-adenoma. As the chief constituent of those tumors included under the fibrous type is the peculiar transparent tissue found around the acinous ducts, the qualifying adjective periductal is prefixed to each,—thus we have a periductal fibroma, a periductal myxoma, and a periductal sarcoma.

It will be observed that sarcoma is included in this classification. It will be described later.

The *periductal fibroma* (Figs. 1 and 2) is an encapsulated tumor varying greatly in size, but as a rule being about as large as an English walnut. These tumors are very movable under the skin and slip freely about when pressed upon by the finger. They can be freely enucleated after their capsule is opened. Their growth is slow and usually painless. In regard to age-incidence, all statistics show that they are most common in persons under thirty. In Williams's cases the mean age was 30.4 years. Of 52 cases which he studied, 28 occurred in women under 30 years.

Treatment consists in removing the growth. For tumors of small or moderate size, excision or resection is the preferable operation, but for very large growths, which, however, rarely occur, amputation of the breast may be required.

The *periductal myxoma* is a tumor the constituents of which are like unto fibroma, although they also contain myxomatous tissue. These tumors appear as rounded or ovoid growths about the size of a hen's egg, being distinctly circumscribed and, as a rule, freely movable under the skin, although occasionally they become adherent and even ulcerate. To my knowledge, retraction of the nipple has not

been observed. Involvement of the axilla, though occurring occasionally, is rare. Treatment is entirely surgical.

In regard to the second or epithelial type, it should first of all be borne in mind that the term cyst-adenoma is applied exclusively to new growths of a cystic character, and not to single or multiple cysts due to involution changes in the mammary gland.

The *fibro-cyst-adenoma* (Fig. 3) is a tumor containing epithelial new growth in a stroma of periductal fibrous tissue, thus differing from the periductal fibroma only in proliferation of epithelial elements. These growths have been called adenomas, tubular adenomas, cystic fibromas, and other equally confusing names. They appear as hard, lobulated tumors, varying in size from a walnut to a large orange, and being freely movable beneath the skin. Upon section they are found to have a definite capsule and a lobular structure containing cysts. From the walls of the cysts outgrowths of connective tissue covered by epithelium are often found. Their growth is very gradual and is not accompanied by pain. They should invariably be removed, however, for the reason that they may become transformed into carcinoma.

In the *papillary cyst-adenoma* (Figs. 4 and 5) there is a proliferation of epithelium in the form of tubes or ducts, the tumor thus resembling the normal structure of the mammary gland. The characteristic feature of the tumors is a papillary outgrowth from the cyst wall, consisting of a connective tissue pedicle, covered with epithelium. They are small, hard, slow of growth, and do not adhere to the skin. Occasionally fluctuation may be detected. The fluid contained within the cysts is usually hemorrhagic. As the growths are often situated just between the nipple the fluid is frequently discharged. Their incidence is most common in married women who have borne children and have reached the climacteric; therefore it would seem that functional activity of the breast predisposes to their development.

Involution Cysts.—In addition to cystic degeneration of sarcoma, fibro-cyst-adenoma, (Figs. 6, 7, and 8) and papillary-cyst-adenoma, which has already been mentioned, single and multiple cysts may be formed during involution of the breast. There is a normal increase of connective tissue during the process of involution, as a result of which the ducts and acini may become compressed and

Fig. 3.—Fibro-cystadenoma. Microscopic appearances. (Warren.)

Fig. 4.—Papillary cystadenoma ("villous papilloma"). Gross appearances. (Warren.)

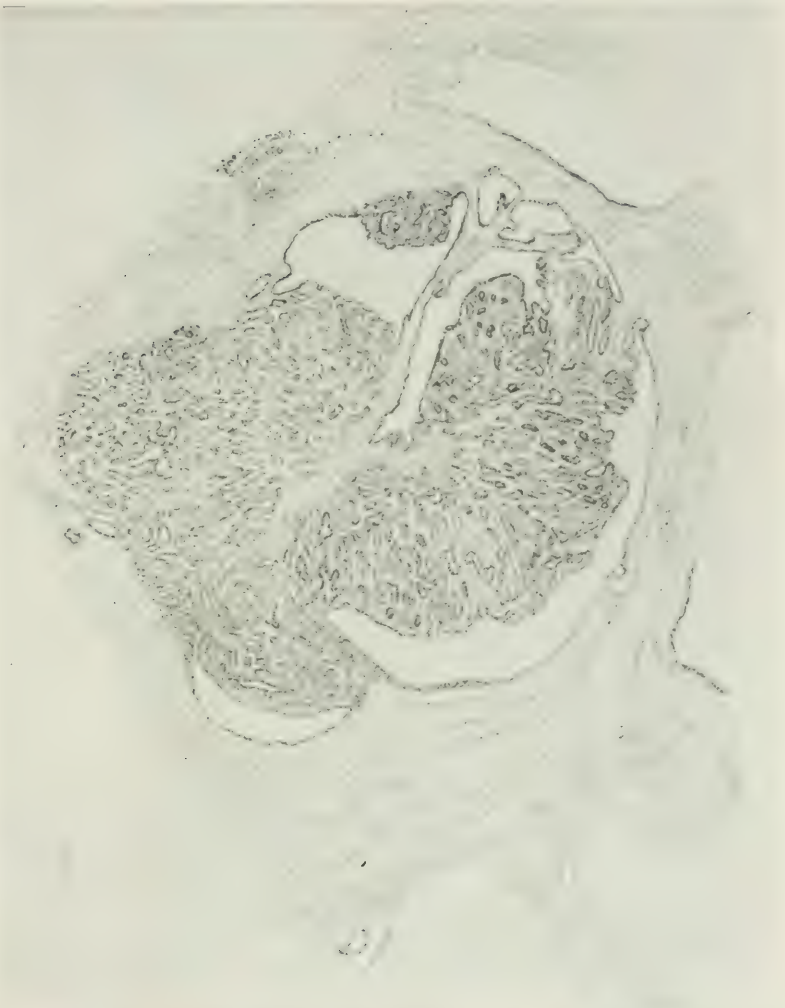

FIG. 5.—Papillary cystadenoma. Microscopic appearances. (Note exuberant growth of epithelium over papillary outgrowths of connective tissue from the wall of the cyst cavity.) (Warren.)

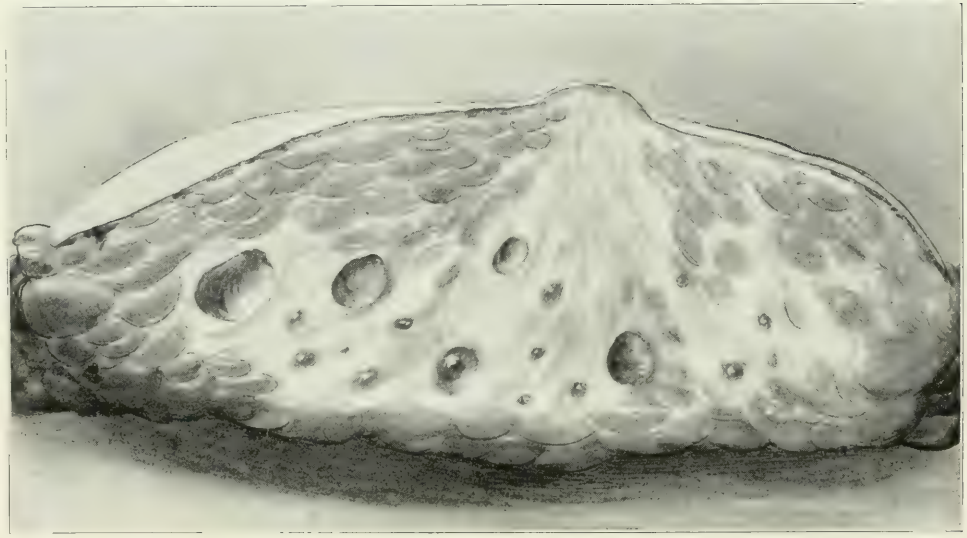

FIG. 6.—Abnormal involution ("chronic cystic disease," "chronic cystic mastitis"). Gross appearances. (Warren.)

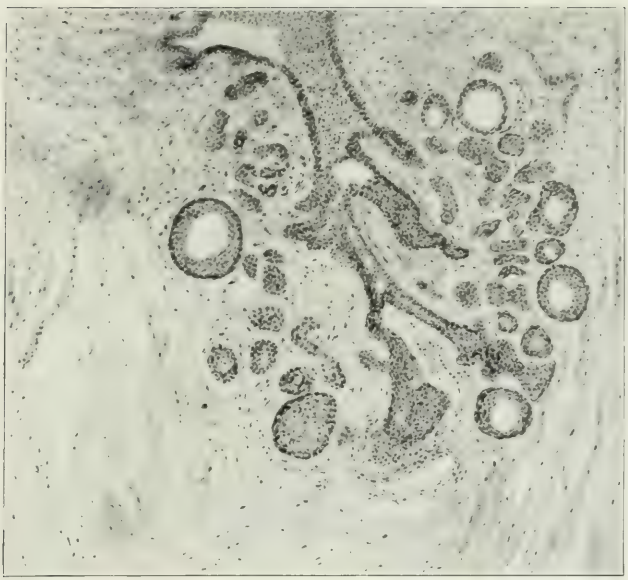

FIG. 7.—Abnormal involution. Microscopic appearances. Acinal type of lepitheial proliferation. (Warren.)

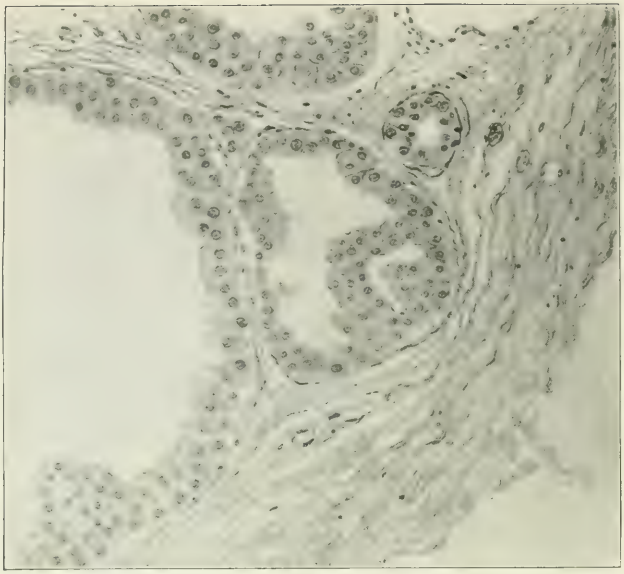

FIG. 8.—Abnormal involution. Microscopic appearances. Papillary and adenomatous types of epithelial proliferation. (Warren.)

FIG. 9.—Abnormal involution and adeno-carcinoma. Microscopic appearances. (Warren.)

obstructed, so that dilatation and consequent cyst formation occurs. This interference with normal conditions often leads to proliferation of the epithelium, which may be so profuse as to constitute malignancy (Fig. 9). Thus out of 30 cases studied by Greenough and Hartwell, there were 3 which showed microscopic evidence of carcinoma. The frequency with which the epithelium in the ducts and acini undergoes proliferation is well illustrated by the statistics of Warren, who found that it occurred in 56 out of 115 cases.

The cysts contain clear serous or dark fluid. They are often multiple, and are most common near the periphery of the breast or near its posterior surface, which rests upon the pectoralis major muscle. The cystic process, however, may be diffused throughout the entire substance of the gland. In size they vary much, some being no larger than a hickory nut, others as big as an apple. They are movable with the breast, but not separately. Pain and tenderness are common symptoms.

Under the microscope some differences are seen which cannot be detected macroscopically. Thus it can be learned by microscopic examination whether the epithelium is proliferating, whether minute papillary outgrowths from the cyst-wall are present, or whether the cysts represent merely a dilatation of the ducts and acini. Cancerous change can of course also be detected in this manner when it is present.

Accurate diagnosis must depend upon microscopic examination, for the more serious of these conditions cannot be distinguished clinically from simple abnormal involution. Several portions of the gland should be turned over to the pathologist, in order that a thorough examination be made.

In regard to the course which these cysts pursue, it may be stated that they sometimes discharge their contents through the nipple, that they may suppurate, or that rupture may occur. They sometimes become stationary in size, but in other instances gradually increase until they involve the entire substance of the gland.

The most rational treatment of these cysts is their complete extirpation. It has already been stated that carcinoma occurs in about 10 per cent. of all cases, and this circumstance furnishes abundant reason for radical interference. I am unalterably opposed to treating them by aspiration, a plan advocated by some.

Formerly it was my practice to cut down upon these cysts and remove them as best I could, or, in cases in which the morbid process had become very extensive, to amputate the breast.

At present, however, I am using the operation first suggested by Gailliard Thomas, and recently elaborated and perfected by Warren, who has termed it plastic resection of the breast. Warren describes this operation practically as follows: The preliminary incision is begun at the lower border of the breast, opposite the middle of the outer arc of the lower inner quadrant, and runs along the lower fold and outer margin to the inner border of the axilla, thus severing the lymphatic connections of the breast with the axillary plexus of lymph-glands. The incision should be carried down to the lower border of the pectoralis major muscle, which should be freely exposed. The dissection is then carried along through the loose connective tissue, which lies between the pectoral facia and the posterior layer of the fascia, in which the mammary gland is contained. With the left hand the operator reflects the breast upward and inward, so that the posterior surface becomes exposed in its entire length. The gland tissue can now be seen through the transparent fascia and easily inspected, and any cysts present readily seen. Usually one or two lie in the same quadrant, which can be removed by a V-shaped incision, without opening the cysts. The apex of the V lies directly under the nipple in the center of the gland. Radiating from this point incisions can be carried into the gland tissue in all directions, exposing and bisecting all small cysts so that none remain which have not been laid open. A second V-shaped incision may occasionally be necessary, but this is rarely the case. The next step after arresting hemorrhage is to close the V incision with two rows of catgut sutures, one along the anterior border and one bringing the posterior edges into contact. The gland is then dropped back onto the pectoral muscle, and it will be found that the various incised portions resume their natural position and fit accurately together. The gland is then anchored to the pectoralis major muscle at the outer edge. Another row of sutures is advisable to hold together the deep layers of the superficial fascia before the outer edges of the wound are closed with silk-worm gut. The buried sutures remove tension from the surface sutures. The dressing is so applied as to produce lateral compression of the two

hemispheres of the breast. I regard the operation of Warren as a most valuable contribution to the surgery of benign growths and have practiced it 3 times in the last month with gratifying results to patients and myself.

SARCOMA OF THE MAMMARY GLAND

Sarcoma of the breast is a comparatively rare affection, constituting not more than 5 per cent. of all mammary tumors. It usually affects young women, although it may occur at any period of life. Gross found it most common between the ages of 30 and 40 years.

In regard to its etiology, little that is definite is known. It is my belief that traumatism plays a more important rôle in its production than in other mammary neoplasms.

All varieties of sarcoma may affect the breast, although the spindle- and round-cell growths are the ones most commonly met with. In some cases both types may be present in the same tumor. Of 156 cases studied by Gross, 68 per cent. were spindle-cell; 27 per cent. round-cell, and 5 per cent. giant-cell.

These growths originate in the periacinous connective tissue, and also from the vessel walls. As the tumor grows, the acini are destroyed by pressure and the ducts become much dilated, forming cyst-like spaces which are interspersed throughout the substance of the gland. Sarcomatous masses may perforate the walls of these dilated ducts, and project into the cavity of the cyst; hence the term cystosarcoma has been applied to this condition.

In the early stages of its development, sarcoma is partly or entirely enveloped by a layer of fibrous tissue resembling a capsule, from which, however, it cannot be enucleated for the reason that the surrounding fibrous layer is continuous with both the stroma of the breast and the tumor itself.

Round-cell sarcoma grows rapidly and not uncommonly proves fatal within three or four months after its onset. With the exception of the very rare melanotic sarcoma, this form is the most deadly that occurs. Spindle-celled tumors are hard, inelastic, and grow more slowly than round-cell ones.

Sarcomas appear as round or ovoid, firm, and rapidly growing tumors in the substance of the breast. They are circumscribed, and

somewhat movable. Ulceration is not uncommon, and, when it occurs, sarcomatous masses are wont to grow into and project from the ulcerated surface. From them hemorrhages may take place. Pain is a variable symptom, being severe in some cases and slight or absent in others. Involvement of the lymph-glands is very rare.

The clinical diagnosis of small sarcomas may be impossible. From carcinomas, these are to be differentiated by the absence of lymphatic involvement, fixation to the skin and retromammary tissues, and retraction of the nipple. Their more rapid growth, and the fact that they occur most frequently in girls or women who have not reached middle life, may also serve as points of differential diagnosis. Finally, when the stage of ulceration is reached the protrusion of the fungous sarcomatous mass will make the condition plain. Fibroma, adenoma, and cystoma, do not grow so rapidly as sarcoma, often being present for months or years without giving rise to local or constitutional disturbances.

Their treatment consists in complete excision. As sarcomas generalize through the blood-vessels rather than the lymph channels, the necessity of clearing out the axilla has been questioned, unless axillary involvement be present. Although it is rare for the axillary glands to be enlarged, as has already been stated, exceptions to the rule do occur, and in view of this fact it is well to remove the glands, especially as the more complete operation will not subject the patient to greater risk and may save her from a recurrence. Regional recurrences are less common than in carcinoma, although metastases to the internal organs are more frequent.

I cannot leave the subject of sarcoma without briefly referring to its deplorable course in pregnant women. I saw recently a woman eight months pregnant, with apparently a large sarcoma growing from the periphery of the axillary quadrant, which was as large as a small cocoa-nut. At the beginning of her pregnancy it was, I am told, no larger than a small walnut. It was wrongly diagnosticated as lipoma. An immediate operation was insisted upon, I preferring to take the chances after a complete operation now rather than to further jeopardize both the woman and child by waiting until after parturition.

I saw another case in consultation during my residence in Louisville, Kentucky, in a woman four months pregnant. The growth

was situated in the upper hemisphere near its periphery, and against my advice the surgeon removed only the growth, leaving the mammary gland. Within a month there was an enormous fungating mass, and the woman died at the end of the sixth month of pregnancy. These two cases, inexpressibly sad as they were, have taught me that early and complete operation should be practiced in every case of a suspected malignant growth in a pregnant woman, as the increased blood supply and amplification of the breast during pregnancy stimulate them to an unwonted growth.

CARCINOMA OF THE MAMMARY GLAND

Of the tumors affecting the breast, carcinoma is by far the most common, constituting about 82 per cent. of all neoplasms. Recent investigations show that it is increasing in frequency, and while this increase may be due partly to a more careful scrutiny of cases, evidence of an indubitable nature has accumulated to show that there is an actual and not merely an apparent increase. Moreover, races which formerly were considered practically immune are now suffering from its ravages. This is especially true of the American negro, as shown by my investigations carried on at the Louisville City Hospital, and also, though in lesser degree, of the North American Indian. Moreover, in addition to its encroachments, racial and geographic, we are finding it far more common in young subjects than was hitherto thought.

Etiology.—To discuss the various theories as to the primary or essential cause of cancer in a paper of this nature would be wandering far afield. I reviewed the subject at some length in the Oration in Surgery delivered before the Minnesota State Medical Society in June 1905.

Precancerous Conditions.—A great deal has been written about the precancerous state, much has been said concerning the malignant transformation of benign growths, and while it undoubtedly occurs in rare instances, though far less often than is generally supposed, a careful investigation reveals the fact that such transformation is usually sarcomatous, and not carcinomatous in nature. In a series of 100 cases of breast cancer carefully analyzed by Warren, in only a single one was there the slightest evidence that a benign growth had undergone cancerous change. Raymond Johnson and others

dissent from the general opinion that breast tumors originally benign become malignant. Certainly in admitting the possibility of such change, we have overestimated the probability of its occurrence.

Paget's Disease.—The belief that eczema, or psoriasis of the nipple and areola, the so-called Paget's disease (Fig.10), frequently results in cancer is not only general but almost universal. In 1895 I questioned the accuracy of this observation, and latterly the most convincing evidence has been brought forward by W. Roger Williams and others.

Of 2,000 cases of Paget's disease carefully analyzed by Williams, in only 0.5 per cent. did cancer develop subsequently. In Warren's 100 cases of cancer, but a single one had been preceded by Paget's disease; thus at most, association between the two conditions is infrequent, perhaps entirely accidental, with the probabilities strongly indicating that the external areola disease is secondary to, and not the cause of duct cancer, the external inflammation being caused by the discharge from a papillary cyst within.

Despite our lack of knowledge of the fundamental and exciting cause of the disease, information relative to its incidence, together with what seems to be predisposing factors, have been recognized by surgeons in all parts of the world. Of predisposing causes, age is the most important, a vast majority of cases occurring in middle life, or about the time of the climacteric. Various statistics, though showing minor differences, are in the main harmonious upon this latter point. Gross's, Williams's and Mahler's analysis of cases all give an average age incidence of forty-eight years, while Sprengel's show fifty as the most common period of its occurrences. It must nevertheless be borne in mind that young women are affected much more frequently than they were formerly believed to be. I have operated upon five cases of cancer of the breast in the last seven years occurring in patients aged 23, 25, 25, 27 and 28 years respectively. All but one—and that in the case of the patient aged 23 years—were typical schirrus growths. McCosh, Richardson, Park, and Warren, have operated on patients aged 19, 21, 22, 22 years respectively. In the Tübingen cases analyzed by Mahler the youngest patient was 26 years. When we recall that S. W. Gross's youngest patient was 28 years, although his experience was large and his ob-

FIG. 10.—So-called Paget's disease. Advanced villous or duct carcinoma of the breast.

servations accurate, we cannot otherwise explain the large number of cases occurring in younger women in the experience of other surgeons.

Traumatism, chronic inflammation, and mammary abscess have often been cited as predisposing causes. The exact etiologic relation, if indeed there be any, which exists between these conditions is not known. It is interesting to note that in 1,000 cases observed by Williams there was only one in which the disease immediately followed injury. Moreover, in 137 women whom he interrogated, only 35, or 25.5 per cent., gave a history of antecedent injury. Per contra, it might be urged that in the remaining 74.5 per cent. a considerable number had received injuries which had passed unnoticed or been forgotten.

Heredity.—Delbet, who analyzed a large number of statistics, finds that the number of cases in which hereditary influence was present varies between 5 and 10 per cent. With the passing of the constitutional theory of cancer, and the demonstration and acceptance of its local origin there began a tendency to minimize the influence of heredity as a predisposing cause. I believe this tendency has been carried much too far. In my own experience, at least one-third of the cases of cancer of the breast that have come to operation at my hands have been preceded by one or more cases of cancer, usually of the breast, in the family. A peculiar soil is inherited, and if the proper seed, whatever it may be, living agent or not, falls upon it, carcinoma may develop.

Recent statistics tend to show that carcinoma of the breast is more common in married women, and especially those who have borne and nursed children, than it is in single women. I cannot agree with those who believe that it is equally common in the single and married, the sterile and fruitful.

Certainly some consideration must be given to such results as have been derived from the examination of hundreds of recorded cases, as for example those analyzed by Guleke, who found that of 982 patients treated in von Bergmann's clinic, 90 per cent. had borne children. As those familiar with the work of the late S. W. Gross may remember, he found that 316 out of 416 women affected with mammary cancer had nursed children.

In view of these and other similar findings, it has been inferred that a direct causative relation exists between the functional activity

of the gland and the occurrence of carcinoma. The periodic changes which take place in the breast as the result of pregnancy and lactation are associated with evolution and involution of tissue, which may predispose to the proliferation of epithelium in the acini, thereby leading to the development of cancer. Perhaps the inflammatory processes to which the gland is often subjected during the period of lactation may lead to the formation of connective tissue which compresses the acini, produces irritation, and thus causes cell-proliferation.

Granted that there be a predisposing causative relation between functional evolution and involution of the mamma, there yet remains a percentage of cases in which this factor can be entirely climinated. Every surgeon of experience has seen cases of cancer of the breast in maiden ladies whose sexual life has beyond any question of doubt been absolutely negative. In such cases it would not be illogical to infer that the organ assumes pathologic activity owing to the fact that it has been deprived of its normal functions.

Fascinating as are such conjectures, and instructive as are some of the inferences derived from the study of large numbers of statistics, they do not by any means constitute a satisfactory explanation for the development of the disease, and I feel constrained to make the same assertion which I made in a paper read over six years ago, namely, that I am as yet an agnostic, denying that any theory yet presented gives entire satisfaction to one viewing the subject from a liberal standpoint.

Pathology.—Several varieties of carcinoma are recognized by pathologists, and although in a paper of this character it would be out of place to enter into a minute description of their structure, the principal forms will be enumerated and their distinctive features outlined. These varieties are the acinous, tubular, scirrhus, and colloid or gelatiniform. Some authors also make a distinction between the gelatiniform and the colloid.

Microscopically the acinous form shows an infiltration of the stroma with round cells. It has a tendency to soften and ulcerate. In the tubular form the process may be even more diffuse than in the acinous, or it may manifest itself as multiple nodules. The microscopic characteristics correspond to the gross morbid anatomy,

Fig. 12.—Cancer en cuirasse.

there being either a diffuse infiltration or localized masses of cells. The scirrhus is comparatively poor in cells. It is characterized by contraction of the stroma and atrophy of the cells. Proliferation, however, continues, but the growth is of slow evolution. In the colloid variety, gelatinous degeneration of the connective tissue surrounding the carcinomatous areas takes place, or the cancerous masses themselves may undergo complete degeneration, so that their original elements no longer exist. In the younger parts of the growth, however, typical cancer cells can be detected.

Symptoms.—The clinical evolution of cancer of the breast is too well known to experienced surgeons to require any detailed description. To the general practitioner, however, who sees comparatively few cases but who as a rule sees these few at an earlier period than it is usually the good fortune of the surgeon to encounter them, an adequate conception of both subjective and objective signs is of paramount importance, inasmuch as the life of the patient depends upon his alertness in diagnosis and the promptness shown in referring such cases to a surgeon.

First of all, it is important to bear in mind that, as a rule, the onset is not marked by subjective symptoms. Contrary to the popular opinion, and one too which I am sorry to say still continues to be entertained by some physicians, mammary carcinoma may attain a well advanced degree of development before it gives rise to much suffering. It is only when the tumor attains a considerable size or compresses nerve-trunks—either of itself or through its associated glandular involvement—that a sense of heaviness and weight in the first instance and of lancinating pains radiating to the arm, shoulder and back, in the second, will be experienced. When ulceration has occurred, of course, more or less pain is found to be present.

The tumor may be discovered accidentally when it is yet quite small, or the patient's attention may not be attracted to it until it has attained a considerable size.

At first it is adherent to the gland with which it is freely movable. It cannot be isolated, as a rule, for the reason that it is intimately connected with the parenchyma of the gland. It is hard and firm, can be readily palpated, and, as the morbid process advances, the tumor becomes adherent to the skin in front and to the pectoral fascia behind. The skin loses its smoothness and softness, and

retraction of the nipple may take place, owing to traction upon the fibrous bands which connect it with the deeper portions of the gland. Gross observed this sign in 108 out of 207 cases, or in a little more than 50 per cent. The nipple may be drawn to one side as well as backwards. Finally, the skin may ulcerate. Not only does the pectoral fascia become affected, but the neoplasm often invades the substance of the pectoralis major muscle itself. Figures 2 to 9 illustrate varying appearances of different types of carcinoma of the breasts.

Dissemination of the disease through the lymphatics is also constantly taking place, with the result that the lymphatic glands which receive the lymph from the channels passing through the breast become diseased. Those of the axilla usually first show signs of involvement, and then those in the subclavian triangle. In a patient whom I recently operated upon two enlarged glands were found between the pectoral muscles. This was the first case in which I had observed enlargement of these glands.

In regard to the exact time at which glandular involvement occurs, statistics show that palpable enlargement may be demonstrated between the eleventh and eighteenth months. Such statistics are of very little value, for the reason that glands may be diseased and yet not be perceptibly enlarged. Much credit is due to Gross, Gussenbauer, Banks, Kermisson and others for calling attention to this fact. It is a matter which the general practitioner should not fail to remember. By bearing it in mind he may be led to refer cases to the surgeon which he might otherwise keep under observation for a longer period. I think it may be safely stated that at least 65 per cent. of all patients show signs of palpable glandular involvement when first seen by the surgeon. As this percentage decreases, the percentage of cures will increase. At operation, however early, there will nearly always be found metastases to the lymph nodes when the axilla is explored. In but a single case have I failed to demonstrate it.

As the disease makes greater and greater inroads, both the local and general condition becomes affected. As already stated, ulceration frequently follows adherence to the skin. The tumor becomes livid and purple, the veins dilated, the skin perforated, and a fissure forms which becomes larger and larger; its edges become everted

Fig. 13.—Large fungating carcinoma of the breast.

and somewhat undermined, although their base is hard and firm. From this surface a purulent, malodorous discharge is given off, and as the ulcerative process encroaches more and more upon the contiguous areas of the breast, hemorrhages may ensue owing to the erosion of vessels. Though usually slight, they may occasionally be so severe as to alarm both patient and physician. The axilla may also ulcerate. Edema of the upper extremity often supervenes, owing to compression of the axillary vein.

With the supervention of such extensive local disturbances the general health manifests signs of impairment. The patient loses flesh and strength, and enters upon a decline which has an invariably fatal progression. It would be futile to portray minutely the clinical picture of a patient suffering with advanced mammary carcinoma, for it is one which unfortunately each and every one of us is still forced to view with only too great frequency.

There is some difference in the mode of evolution and duration of the various forms of cancer of the breast which have been mentioned, and it may not be amiss to call attention to these differences. Thus acute cancer, so called, pursues a rapidly fatal course, frequently terminating in death within a few months after its onset; so too the softer varieties, the so-called encephaloid growths, are more rapid in their progress than scirrhus. In the former the breast may attain twice or thrice its natural size within a few months. In the gelatiniform or colloid variety the tumor may be so soft that it seems to fluctuate. Ulceration and axillary involvement also take place earlier in these forms than in scirrhus.

Diagnosis.—No one will question that better results in the future, if they are to be had, must follow earlier rather than more extensive operations. It is desirable, as Sir William Banks pointed out more than twenty-five years ago, to operate on a cancer of the breast when it is no larger than a pea, if patients would apply to us at that time or if diagnosis could be made. Unfortunately, things have improved but little, and general practitioners do not send us their cases much sooner than they did then, owing partly to their inability to make a correct diagnosis, but, also, and in equal degree no doubt, because of their lack of faith in the power of surgery to effect a cure.

Pessimism is so easy, optimism so difficult, when dealing with

cancer, that medical men can hardly be blamed for quoting the false teachings of a past era. Rarely a month passes that the unfortunate experience and still more regretable teachings of a late illustrious professor of surgery in Philadelphia, are not quoted to me by some of his pupils. It is passing strange that the more advanced and better teaching of a contemporary are not more generally remembered, for it was there that the complete operation and teachings of Moore of London were first practiced and inculcated in America.

While the age of the patient and the presence of a hard nodulated growth the development of which has been slow and painless, will naturally excite suspicion of carcinoma, these factors will not always suffice to make a positive clinical diagnosis. Age, of course, is of great value, the disease occurring in the majority of instances at about the menopause, but, as has already been stated, it is by no means so rare in young women as it was formerly thought to be. Although the majority of hard tumors of the breast occurring in women before the thirtieth year will be found to be adenomas or fibromas, still the possibility of their being carcinomas must be remembered. Soft, rapidly growing tumors in young women and girls usually prove to be medullary sarcomas, whereas very large tumors are mostly cysts, fibromas, adenomas, or sarcomas.

The location of a tumor is of great significance from a diagnostic standpoint. Malignant growths are most frequently found in the upper and outer quadrant, lower or axillary quadrant, and behind the nipple. Benign growths, on the contrary, affect the upper and inner quadrant, or sternal half of the gland. While retraction of the nipple is occasionally seen in benign growths, it is rare, occurring in only from 3 to 6 per cent. of such cases; it is merely accidental and usually of congenital origin. Benign tumors are also movable from beginning to end. They are distinctly encapsulated, and slip under the finger like a marble under a cloth. In benign tumors the skin is not affected. Axillary involvement is present in only 3 per cent. of all cases, and is then of course irritative, not metastatic.

The history of heredity, trauma, mastitis, discharge from the nipple, are of little if any value, as they are about as frequently obtained in benign, as they are in malignant affections. When a cyst is suspected, aspiration settles the question.

FIG. 14.—Bilateral mammary carcinoma.

FIG. 15.—Carcinoma of the right mamma, beginning in the axillary quadrant of the breast.

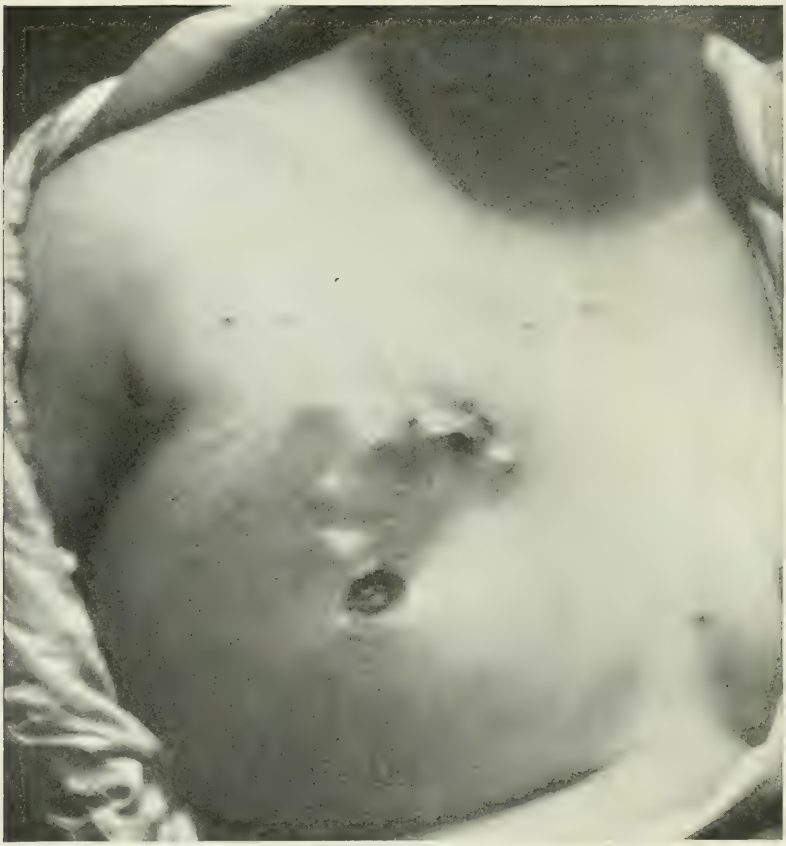

FIG. 16.—Typical scirrhus carcinoma of the upper inner quadrant of the breast.

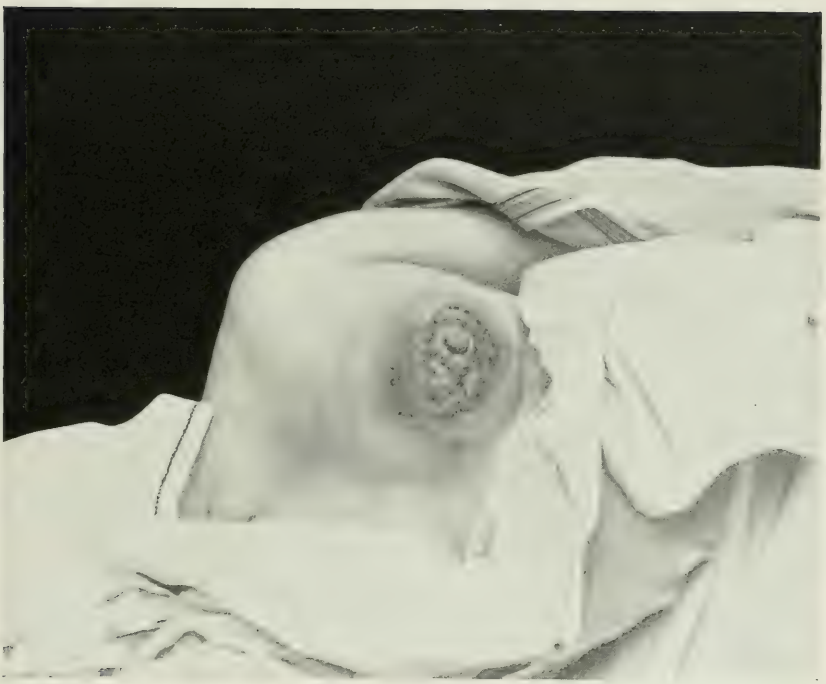

FIG. 17.—Advanced carcinoma of the breast.

FIG. 18.—Inoperable carcinoma of the breast.

FIG. 19.—Recurrent carcinoma of the breast.

In all doubtful cases there is a conservative, and, I believe, a safe course to follow. Instead of making, as many advise, an incision into the growth for diagnostic purposes, and then closing the wound—a practice not altogether satisfactory, and certainly not free from danger—I prepare for and get the consent of the patient for a complete operation. A competent microscopist is present with his apparatus, prepared for rapid yet accurate work. If the chances stongly favor benignity, the tumor and its capsule only are removed and submitted for an immediate examination and opinion.

Usually, in less than ten minutes the report is returned. If there is a strong suspicion of malignancy, a portion of the growth near the center is removed, the wound at once plugged with gauze and nothing further done until the pathologist reports. If the piece thus removed proves to be malignant, the complete operation is immediately performed. I have followed this practice for twelve years and have never known a mistake to be made in diagnosis, nor been made to suspect that harm had resulted from the liberation of cancer-cells which affected adjacent healthy tissue. I think, however, that plugging the wound made in the growth is important, and that the wound thus treated should be left entirely alone until the breast and the muscles have been removed. Therefore, it would seem that the danger can be minimized if not actually prevented, and the slight risk incurred is as nothing to the definite information gained. I can point to at least half a dozen women, happily married and with practically perfect breasts, where everything might have, probably would have, been different had not this precaution been taken in operations for benign growths before marriage. Further, I have been prevented from mistakes in at least two malignant tumors. When operating apart from a well-appointed hospital and its pathologist, the macroscopic appearance of the tumor, its color, consistence, resistance to the knife, and, above all, the presence or absence of a capsule, will as a rule prevent a mistake in diagnosis. Stiles's nitric-acid test may be applied to the tumor with advantage. Every growth removed from the mammary gland should of course be examined with the microscope as soon as practicable.

Prognosis.—At the present day it hardly need be said that the earlier the operation can be performed the greater are the chances

of recovery. This fact was well attested by the statistics of some of the older surgeons whose observations were recorded before the more thorough methods of operating had come into vogue. Thus, Winniwarter found the average duration of life after operation in cases in which the operation was done before gross involvement of the axillary or supraclavicular glands were involved, to be 22 months, whereas in those which had such glandular involvement it was only 13 months. Naturally much depends upon the thoroughness with which the diseased structures are removed. It is most gratifying to note the greater percentage of patients who remain free at the end of 3 years since the extensive operative methods practiced by Halsted, Meyer, Stiles, Keen, Lennander, myself, and others have come to be more generally employed. Although the results obtained by different operators show considerable variation, the highest percentage of cases remaining free from recurrence at the end of three years is attained by those surgeons who do the complete operation. This is a percentage of 50, and should serve as a conclusive argument in favor of the extensive removal of all accessible tissue which can possibly be diseased.

Volkmann's 3-year limit should be extended to at least 5 years, as careful investigations show that recurrences after this period are not uncommon; some have been reported after ten years, or even longer.

In regard to operative mortality, my statistics, based on 2133 operations performed since 1893, by twenty-one American surgeons, show the deaths to be less than 1 per cent. This seems almost incredible when contrasted with the 15 to 25 per cent. mortality for incomplete operations in pre-antiseptic days.

Carcinoma of the breast pursues a more fatal course in young persons than in old, for the reason that in the former class the lymphatics are more numerous and more patent.

Treatment.—To operate at the earliest possible moment and remove every particle of accessible tissue which is likely to be infected, is a rule which I believe should be followed in its entirety with very few exceptions. The pectoral muscles, both major and minor, should always be sacrificed, so that the axilla can be thoroughly cleared out and all diseased tissues removed in one mass, thereby obviating the danger of contaminating the wound with

cancer-cells by dividing diseased structures, and also for the purpose of preventing shreds of cancerous tissue remaining behind, an occurrence which might readily happen if a piecemeal dissection were made. In view of the fact that there is a direct communication between the accessory lymph channels of the breast and the supraclavicular glands, the subclavian triangle should be explored *in all cases* in which the cancer is situated *superiorly* and near the margin of the breast. Of course there is no question as to their removal when they are palpably enlarged, provided that the case is considered operable.

In regard to the operative technic, it is my belief that the incision should be so fashioned as to include the skin over the entire breast, thoroughly exposing the pectoral muscles from origin to insertion and completely uncovering the axilla. I prefer to remove the muscles from insertion to origin instead of from origin to insertion, for the reason that the axilla is uncovered at once and can be cleared from above downward, thus insuring a more complete dissection and facilitating the removal of all diseased tissue in one mass. Moreover, the large heavy mass containing the breast is not in the way while the axillary dissection is being made. Hemorrhage is also more easily controlled by reserving Halsted's technic.

Many excellent incisions have been devised, but I am of the opinion that the one first practiced by Professor Warren will be found the most useful in the majority of cases. It insures primary union after the removal of extremely large growths, and when modified by making a second inverted Y above as well as below—a modification which I have introduced and practiced in many cases—permits even more extensive removal of the skin than would otherwise be possible. In cutting the flaps the skin is undermined according to Shrady's method, and deep relaxation sutures are used in addition to those affecting approximation.

Drainage should always be employed. Unless it be used considerable time will be consumed in arresting oozing from minute vessels, which while unimportant as concerns loss of blood, may give rise to the formation of a clot, and possibly lead to suppuration.

It is now generally known, especially among surgeons, that these extensive operations are not followed by much interference with the arm. In all of my cases its movements have been fairly well pre-

served. My patients have been able to feed themselves, adjust their clothing and even comb their back hair, which is a crucial test and most difficult to accomplish when there is much restriction in the latitude of motion. W. J. Taylor states that one of his patients with both breasts removed, as well as both pectoral muscles on each side, has made her living for a number of years cutting out clothing.

Wounds of the axillary vessels have been unusual since both pectoral muscles have come to be more frequently removed. It is interesting to note that of 24 intentional resections of the axillary vein, not one was fatal. Moreover, the edema following was inconstant and transitory, and was never very troublesome.

JACKSONIAN EPILEPSY DUE TO BRAIN TUMOR; OPERATION; RECOVERY. TUBERCULOUS ABSCESS OF THE SHOULDER-JOINT, WITH EMACIATION AND MUSCULAR ATROPHY, MISTAKEN FOR PROGRESSIVE MUSCULAR ATROPHY

A CLINICAL LECTURE DELIVERED AT THE PRESBYTERIAN HOSPITAL, NEW YORK

BY MAX SCHLAPP, M.D.

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Gentlemen: The first patient that I have to show you to-day has this advantage over the ordinary patients that come to a neurologic clinic—he is cured. It is almost a general rule that the more interesting the neurologic case the less amenable it is to treatment, at least as regards hope of ultimate complete cure. This young man insists that he is now perfectly well, and he is certainly able to do his work quite as well as ever; he has no awkwardness in his movements, and yet two years ago, he dragged his leg very sadly and his left arm was practically useless. In the meantime he has had an operation involving no little disturbance of his cerebrum; yet all is well.

It is sometimes said that operations for brain tumor are not often followed by success. There is a certain amount of truth in this dictum, but when the localizing symptoms have been such as to enable the neurologist to point out to the surgeon just where operation should be done, and when the tumor found is not malignant, the prognosis is very often quite favorable. In this present case it would have been temerity to promise complete recovery; this has taken place, however, and that too in spite of the fact that almost complete disability had existed for many months, and symptoms had extended over eight years. Fortunately the form of tumor was that which yields most readily to treatment, and very seldom gives rise to recurrence. In these cases, especially

in young patients, the rule must be to operate, since it presents the only hope of relief.

Just three years ago this young man, then 19 years of age, came to the nervous dispensary of the Presbyterian Hospital complaining of weakness in his left arm and leg. He had been employed as a grocer's boy for some years, until the increasing weakness of his arm and leg made it impossible for him to continue at his occupation. When he came under observation he gave a history of having had, about a year previously, certain convulsive seizures on the left side of his body, especially in his left arm and the corresponding side of his face. The description of these indicated that he had suffered from true Jacksonian epilepsy. Further inquiry seemed to show that his condition was very probably traceable to an injury received about five years before his first symptoms were noticed.

His family history was negative both as regards tuberculosis and neoplasms. His own previous history, with the exception of the injury to his head, had very little that was of interest before the beginning of his present illness. He had whooping cough as a child, but without any serious sequels. He had never used alcohol to excess nor, indeed, indulged in it to any extent, and he denied venereal infection. His mother said that he had suffered from malaria and rheumatism, but the symptoms she gave of these diseases made it very dubious whether they had really been present.

The most interesting and important item of his history is that eight years ago, while working on a new building, a barrel fell from the sidewalk above his head, a height of about five feet, and struck him just above and behind the right ear. He did not lose consciousness at the time, and refused to permit the doctor whom he visited after the accident to put any stitches in the scalp wound which occurred at the time. The wound healed perfectly, leaving scarcely any trace except a linear scar on the scalp, and until very recently he has had no further symptoms pointing to any serious injury of the head. Some three years after the accident, just after going to bed one night, a sharp pain, which began in his left hand, crept up his arm to his shoulder, while at the same time there was a twitching of his fingers. After a time his wrist and then his arm and shoulder were drawn up,

and finally he experienced pain and a sensation as though his face on the left side were contracted. After this he lost consciousness, and remained unconscious for nearly a half hour.

After awaking he says he felt perfectly well. On closer questioning he confesses that there was a tired feeling, but of this he did not take much account. He is one of those phlegmatic individuals who do not notice pain or discomfort very much, and the only thing that bothered him really was the weakness of muscles which began to develop. After this first seizure there was no paralysis, stiffness, or pain in his arm or face. Just before he became unconscious, however, he thinks he remembers that the spasm became very severe, and his whole arm was stiff,—that is, was in firm tonic contraction. Occasionally after this he had slight pain in his wrist, arm, and shoulder, with some muscular spasm. These came on in attacks, which recurred more and more frequently. At the end of two weeks they were recurring nearly every day, though they did not produce any special disability.

After two or three months, while sitting quietly in his chair at work, he had an attack exactly similar to the first one. This attack lasted only fifteen minutes. On waking, he walked home, a distance of several blocks, without delay and without discomfort, though he felt weak on his left side and limped at first, but only for a few minutes, when he regained his strength entirely. For a month after this, except for the slight twinges of pain with occasional twitchings, which recurred every day or two, he had no further symptoms.

Then one day, while pushing a grocer's cart, he felt the pain and spasm beginning in his right arm, and realizing that it was severer than usual, and that he was going to have one of his attacks, he ran toward a stoop to sit down. Before he got there, however, he says himself, "he began to go sideways, bumped into a wall and fell down," losing his senses. This attack lasted more than half an hour. His left side felt weak after this, his left leg dragged a little, and he staggered toward this side. These symptoms lasted, however, only a short time. Then he was able to go on with his work again without bother.

This attack occurred nearly six years before he applied at

the Presbyterian Hospital for treatment, and he has not had a similar attack since. He had slight attacks of pain and twitching for some time afterward, but these gradually grew less noticeable and less frequent, and stopped entirely before he came under observation. For the three months before he came under observation, however, a new set of symptoms developed, and he and his friends noticed that he moved his left leg awkwardly, and with a decided limp. His arm has been growing gradually weaker, until it became so paralyzed as to be useless.

About two months before his application to the dispensary he suffered from an attack of very severe headache. It came on at night when he was asleep, without known cause of any kind, grew steadily worse until midnight, when he suddenly vomited violently. After vomiting the headache became somewhat better, and entirely disappeared before the next morning. In describing it he said that at first the pain was all over the head, and as it grew more severe, it seemed like an iron band about his head. After the vomiting he felt the pain almost exclusively in the temples and eye-balls. A week later he had another bad headache of the same kind. This came on about the same time at night. He vomited about midnight once more, and the pain in his head left him in the early morning. Six weeks later there was a third attack of headache, just like the other two, only that it continued to grow worse until eight in the morning, when he vomited, and then the headache disappeared within a short time.

This was his history when he came under our observation, asking for treatment for his headache, for the weakness of his left leg and for almost complete inability to use his left arm. We found him well nourished, not at all anemic, and his tongue, moist and clean, was protruded in the median line, though there were some fibrillary tremors present in it. His post cervical lymphatic glands were enlarged, his pulse full, soft, and regular, his heart normal in size and action, but with a faint blowing systolic murmur at the apex. Careful physical examination of all his other organs showed no abnormalities except in his nervous system.

His pupils were equal and reacted to light and in accommodation. There was some evidence of facial paralysis on the left

side. He talked somewhat thickly, but there was no alteration in his speech. His left arm was almost completely paralyzed, though there was some slight power in the shoulder muscles. The arm was quite rigid in adduction and the forearm was flexed. The muscles were soft and flabby, but evidently only from disuse, as they showed practically no wasting. The muscle and tendon reflexes in the arm were increased. Whatever muscular atrophy was present was more marked in the forearm. His left leg showed distinct loss of power, though he could move it in every direction. The muscle and tendon reflexes in this limb were exaggerated and Babinski's sign was present. Ankle clonus could be elicited without difficulty.

Careful examination of his eye-grounds was made and marked optic neuritis was found in each eye; the vessels were tortuous and dilated and there was choroidal atrophy in the region of the nerve-ending. The optic disc in each eye was swollen about two millimeters. In general, the pathologic conditions present were found to exist to about the same degree in each eye.

Some of the most important symptoms consisted of sensory disturbances in the affected arm and hand. The deep muscular disturbance was very marked; he could not tell the positions of his fingers, and he was not able to state with any confidence the position of his hand or the amount of angular movement in his elbow. The tactile sense in his fingers was extremely blunted, and below his elbow was distinctly less than on the other side. Pain and temperature senses were not disturbed. There was distinct astereognosis, and such familiar objects as a pencil, a knife or a coin could not be distinguished when placed in his hand.

A diagnosis of intra-cranial neoplasm was made, the reasons therefor being the characteristically developing symptomatic picture, to be traced in the history and the present condition; the original injury followed after some years by Jacksonian epilepsy, with distinctly focal symptoms; later severe attacks of pain, followed by vomiting without special reason on the part of the digestive tract; the gradually developing weakness of the arm and leg, with some involvement of the face; the choked discs and the sensory disturbances,—all pointed distinctly to the existence of an intra-cranial tumor. In so young a patient it seemed emi-

nently advisable to try what could be accomplished by operation.

The outlook after operation depended, however, to a very great extent on the nature of the tumor that might be present. The very slow growth, taking years for its increase to such a size as to produce symptoms, pointed to a glioma, or a very slowly forming cyst. The injury might have given rise to scar tissue, the irritation of which would eventually produce gliomatous growth, but it was more likely to have produced cyst formation, and it was hoped that this was the pathologic condition that would be found, since the prognosis after the removal of a cyst from the cerebrum is much better than that of a glioma, and is, indeed, as a rule, very favorable.

This tumor was evidently situated in the posterior central convolution back of the second frontal convolution, not exactly in but in the neighborhood of the arm and leg centers, and involved them indirectly by pressure, rather than directly.

Accordingly, the patient was referred to the surgical wards of the Presbyterian Hospital, where he was operated upon by Dr. Wolsey. The usual incision for the examination of the area near the fissure of Rolando was made, and a horseshoe-shaped flap was turned down. As soon as the flap of skull was lifted, the brain bulged through the opening somewhat above the level of the skull, though still, of course, covered by dura and retained by that membrane. The color of the bulging part was rather yellow, and the usual pulsation noted when normal brain substance is exposed was not present. An incision was made into the dura, and the brain bulged still further through it at the base of the flap, gradually becoming bluish in color. Shortly after the incision in the dura the bulging brain tissue ruptured, discharging a stream of yellowish, slightly turbid fluid, about four or five drams in amount, which spurted some eighteen inches high. This was found to come from a cyst close under the surface of the brain. After this evacuation of the cyst, the brain began to pulsate. The flap of dura was then elevated, bleeding points in the brain tissue were secured with fine catgut on a curved needle, and the opening in the cyst wall enlarged and the cyst explored with the finger. It was found to be two inches long, oval in shape, and contained within it, a pedunculated, nodular tumor of medium con-

sistency, more or less spherical, about one and a quarter inches in diameter, and attached to the inner cyst wall at the upper posterior portion. After enlarging the opening, the tumor was easily removed, shelling out without noticeable bleeding or laceration of tissues.

The etiology of the cyst thus removed is not difficult to understand. The severe injury to the head ten years ago, caused either the rupture of a small artery with hemorrhage, though not of large size, or caused thrombosis and interruption of the circulation to a small portion of the subcortical region, with consequent softening. This of itself, was not large enough at the time to produce any focal symptoms.

Following the softening of the tissue there must have been a proliferation of the connective tissue from around the blood vessels. This process eventually led to the formation of the wall of fibrous tissue about the area which had become softened, separating it completely from the normal brain tissue in the neighborhood. In time the broken down tissue within the cyst, the detritus of degenerate cells was carried away by fat granule cells and epithelioid cells, leaving the fluid in the enclosed connective tissue cavity quite clear. From the fibrous tissue wall the tumor in the case developed. In the course of its development the pressure within the cyst was gradually increased until pressure symptoms became noticeable.

That this cyst was not of congenital formation, due to an anomalous pinching off of some portion of the lateral ventricle, a not very unusual incident, or to some other malformations of the ventricles during embryonic life, is clear from the fact that in that case it would have been lined with ependyma cells. The existence of a fibrous tissue tumor within the cyst showed that it was not ependymous in origin, since a tumor springing from that sort of tissue would have been a glioma. As can be seen from a microscopic section of the tumor it is true fibroma, a connective tissue tumor, and it very likely developed from the cyst wall after this had been already formed. The cortex of the brain was intact about the tumor, so that it was not an ingrowth from the pia.

The tumor was connected at one portion of the lining of the cyst and derived its nourishment, being rather freely supplied

with blood vessels, from this part. The tumor was not of very firm consistency, but was distinctly fibromatous in character, and even appeared so to the naked eye on section.

Three weeks after the operation the patient was allowed to get up, and was able to walk quite well, though there was some incoördination of movements in his left leg. At this time he was able to recognize the position of his left thumb and first finger when they were moved with the eyes closed, though his muscular sense in the other three fingers was still imperfect. The Babinski sign was still present.

When he was discharged, just four weeks after the operation, the general condition was good, there was no sign of facial paralysis, and the motions of his left arm and leg were practically normal, though the muscles were weaker than in the right arm and leg. The muscles had grown much stronger than they were before the operation, but had still not regained their normal size. The wound in the skull had closed with the exception of a small sinus not more than a centimeter in depth. The bone flap had united very thoroughly and there was no depression in the skull.

The patient has been seen within the last few weeks and is now driving a brick wagon, and says he feels absolutely no inconvenience from the rather serious experience through which he passed. He is able to use his hands with equal force, and none of his muscles seem to be lame. He never suffers from headache, and has no vomiting and no nausea.

There seems no reason to doubt his complete cure, from his appearance, or from his gait, or from what fellow workmen say of his capability for using his hands and legs. This would seem to be one of those very fortunate cases by which, notwithstanding the existence of very serious symptoms due to a pathologic process extending over eight years, operation led to the removal of the tumor before any serious damage had been inflicted upon the nervous system. It illustrates how much may be accomplished by localizing diagnosis and recourse to surgery even in apparently hopeless cases, for without operation this young man would have been left incapable of earning his livelihood for the rest of his days, the span of which, moreover, would not have been very long. With the gradual increase of intra-cranial pressure that had taken place

FIG. 1.—Tuberculosis of the shoulder-joint simulating muscular atrophy. *a*, Abscess originating in the joint.

during the last year, it would not have been a question of much time before a fatal termination would have been imminent, because of interference with important vital functions. The complete palsy which had existed for some time in the arm and leg might have discouraged the idea of operation, because of the fear that degeneration of nerve fibers had already taken place, and the eventual outcome would not be favorable. The patient has, however, been left practically without a mark of his previous illness.

Case II.—I have here a photograph (Fig. 1) which is of interest because it recalls very well the details of a case in which two mistakes in diagnosis, that are not uncommon, were made before the patient came to the outdoor service of the Presbyterian Hospital. He was a man, about 35 years of age, who had lost considerable in weight, had suffered from some vague pains in his shoulder, at first thought to be rheumatism, and had traveled in Europe with the idea that change of scene might enable him to get into better constitutional condition. He did not improve much, however, and on the return trip on the steamer a slight strain of his left arm gave rise to acute symptoms of pain and a distinct inflammatory reaction in his shoulder. The weather had been raw for several days, and the ship's surgeon pronounced the disorder to be rheumatism, and put the patient's arm in a sling. He did not improve, however, but rather grew worse.

After he had been about four weeks in New York, having lost considerably more in weight than before, he called on a physician who after seeing his emaciation, and especially the marked atrophy of the muscles around his left shoulder, told him that he had very probably progressive muscular atrophy. He gave him a note to this effect and sent him to the dispensary of the Presbyterian Hospital. Because of this diagnosis, he was referred to the neurologic service, where the following notes of his condition were made:

The patient is very much emaciated, and there is especially around the left shoulder joint marked atrophy of all the muscles in any way involved in the motions of the joint. The deltoid is so much atrophied as to reveal the outlines of the shoulder-joint very completely. The supraspinatus and infraspinatus muscles are both markedly affected in the same way. Even the trapezius

muscle as far as regards the fibers that run toward the shoulder is markedly atrophied. Besides this it was noted that there was loss of knee jerks, though the reflexes in his arm muscles below the elbows were present on both sides. On the other hand, there were no fibrillary twitchings, no sensory disturbances, and no reactions of degeneration.

His general condition, however, aroused suspicion of some inflammatory process at work. His pulse rate was above a hundred, and his temperature was 100° F. He gave a history of suffering from sweating rather easily, especially at night, and a feeling of intense fatigue in the afternoon if he moved about much. He presented just below the shoulder-joint, as can be seen in the photograph, a distinct enlargement in the course of the biceps muscle. This had been considered to be some anomalous muscular tumor by the physician who examined him previously, but on careful palpation, fluctuation was demonstrated. This seemed to settle the diagnosis that some form of abscess had occurred in the upper arm, perhaps in the joint itself, and the fluid material had found its way thus far down within the sheath of the biceps muscle. As a matter of fact, when the abscess was opened, it proved to be what the Germans call a "senkungsabscess," that is, a gravity collection of pus, finding its way downward from an affected joint. At the examination some indistinct grating could be felt in the joint surface.

The interesting points about the case are the absence of the knee jerks, and the atrophic condition of the muscles. In my own experience I have found not infrequently, in severe cases of tuberculosis, especially when there is much emaciation, that the knee jerks are absent, or can only be elicited after the exercise of the greatest care. In three recent cases I am sure that they were absolutely absent. With regard to the atrophy of muscles around the shoulder joint, it must be borne in mind that whenever a joint is affected, all the muscles around it become atrophied by a sympathetic reflex. This is due not only to the lack of use of the muscles around the joint, but to a distinct lessening of the trophic influences on which the nutrition of these muscles depends. It would seem as though nature's conservative mechanism is so coördinated that

when a joint is affected so badly as to need rest, all the muscles in the neighborhood are refused the trophic impulses that ordinarily enable them to do their work, and they thus become incapable to a great degree of accomplishing movements of the joint. As a matter of fact this atrophy of muscles around a joint is one of nature's most wonderful conservative processes, and directly curative in effect if not interfered with. It must not be mistaken, however, as in this case and in many others for some distinct pathologic process.

THE PRINCIPLES OF THE TREATMENT OF FRACTURES, WITH A SPECIAL CONSIDERATION OF THOSE OF THE UPPER EXTREMITY

BY GEORGE G. ROSS, M.D.

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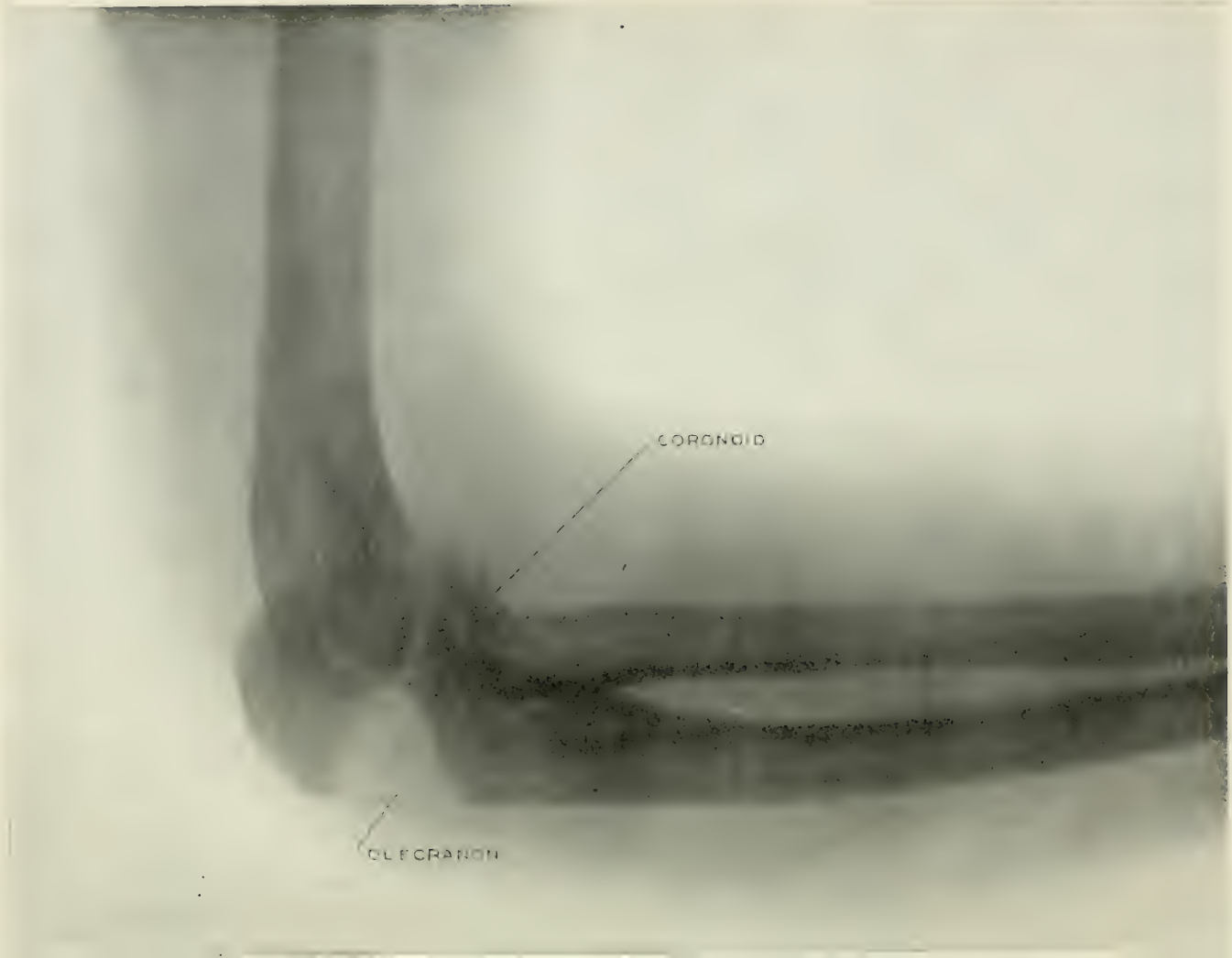
The influence of the systematic corroboration of clinical knowledge of fracture by means of x-ray photography has been exerted upon treatment through accurate diagnosis and knowledge of the pathologic condition existing in a given fracture or class of fractures. Our present knowledge of the pathologic anatomy of fractures enables us to apply the principles of treatment to a better advantage and to obtain better results than were obtained prior to the discovery of the x-rays. In the rarer forms of fracture, especially those of minor anatomic degree about the joints, the x-ray has established a line of treatment that, while along the line of recognized rules, has entirely changed the prognosis of these cases. The ability to diagnose correctly and to confirm the direction of the line of fracture, particularly the degree and the relation of the fragments to each other, has enabled us to correct deformities that were underestimated or not observed, and therefore incompletely reduced, resulting in degrees of lost function that to-day would be a lasting reproach upon the surgeon. The estimation of pathologic conditions has been materially improved by the sound knowledge of the normal anatomy that has been partially worked out by the German profession, and in this country by such men as Hickey.

The axiom that restoration of function is more important than restoration of contour in the affected part has been strengthened until it is now an established scientific belief. True it is that function is sure to follow the complete reduction of deformity in the vast majority of cases; but this is not always possible, as the following case will illustrate:

Mr. L., aged 45 years, sustained a fracture of the olecranon and the coronoid process of the ulna of the left elbow. (Figs. 1

FIG. 1.—Fracture of the olecranon and coronoid processes.


CORONOID
OLECRANON

and 2.) The principles of treatment for these two fractures are diametrically opposed. The olecranon fracture should be treated in full extension; the coronoid, in half flexion or at a right angle. The treatment consisted in full extension for two weeks, so that a sufficient amount of callus could be thrown out to insure strong fibrous union with a minimum amount of displacement. The forearm was then gradually brought to a right angle with the arm and thus retained for two weeks longer, the period of fixation being five weeks. Passive motion was commenced on the seventh day, and was continued with gentle massage. The result was functionally perfect, although there was a separation of the olecranon fragments of a full half-inch.

In the vast majority of fractures, prolonged fixation is not necessary or desirable. The fracture should be reduced, for the patient's comfort, as soon after the accident as is possible. A fixed dressing should be applied and retained until the swelling, soreness, and muscular contraction have disappeared, after which systematic, skilful, and frequent dressings should be the rule. By this line of treatment, atrophy of the muscles and contractures are minimized; and the venous stasis and consequent swelling of the limb, so common after prolonged fixation, is prevented. By skilful and gentle manipulating, the injured member can be dressed, washed, and massaged; and if desirable, passive and even active motion may be executed, without endangering the integrity of the union at the seat of fracture.

The average time of fixation is from four to five weeks; but the time will vary with the character, extent, and location of the lesion. Oblique fractures of the long bones require a longer period of fixation than do transverse fractures. Fractures near or involving a joint should not be kept in a fixed position longer than four weeks, and should be systematically and regularly treated by passive and active motion. In sprained fracture about joints, three weeks will suffice for firm union.

The belief in treatment by immediate operation, with fixation of the fractured ends by wiring, plating, or pegging, has been steadily gaining ground in recent years. There are a few fractures in which the operative procedure is indicated, notably fractures of the patella. Here all forms of conservative treatment fail to secure

satisfactory bony union, and the open operation is the wise course to pursue—provided the operation is performed by a skilful surgeon and in rigid antiseptic circumstances. The methods of fixing the fragments are wiring the fragments and suturing the torn capsules. The removal of the blood and exudate, the removal of fringes of the torn capsule from between the fragments, and the accurate apposition and fixation, in the presence of asepsis, insure firm bony union without deformity, and complete restoration of the joint-function. The operation should be deferred for a week or ten days, so that the maximum swelling may be attained, and in order to lessen the high value of the culture-medium offered by the blood-serum exudate. In fractures of the long bones in which reduction of the fragments cannot be accomplished, or when, after reduction, the deformity tends to recur, and when function is threatened, open operation is indicated. Nothing is to be gained by operating within the first seven or eight days.

The difference in treating fractures of the extremities lies largely in the fact that fractures of the upper extremity are ambulatory, while the majority of those of the lower are bed-cases. The rule of procedure in either case is to reduce the fracture by extension, counter-extension, rotation, and manipulation; and to keep the fracture reduced by fixation until the process of repair is sufficiently advanced to insure permanent healing. The method of fixation is largely a matter of personal preference, depending on early teaching and experience. The old-fashioned wooden board-splint—adapted, as it can be, to nearly every kind of fracture of the extremities, and always being at hand, or readily made by the nearest carpenter or by the surgeon himself—has not been entirely displaced by the plaster-cast, metallic splint, felt splint or binder's board. The use of plaster of Paris as a dressing for fractures is of the greatest value. The plaster-cast, when properly applied, is safe and efficacious; but in unskilful or inexperienced hands, it may become dangerous and worse than useless.

Operative treatment is to be considered under the headings of immediate and remote operation.

Immediate operation is indicated in comparatively few cases of fracture of the extremity. In all compound fractures, immediate operation is to be considered; because it permits of a thorough

cleansing of the injured area, not only on the surface of the limb, but also in the deeper involved structures. We can fix the fragments permanently by wiring, plating, pegging, and sewing with heavy chromic catgut. In comminuted fractures and compound comminuted fractures, operation is indicated, for the same reason as that just given; and also because the opportunity to remove loose or detached fragments, and to control the bleeding and establish drainage, is afforded.

In simple fractures that are uncomplicated, open operation is contraindicated; because it is unnecessary, adds to the risk of infection, does not shorten the time of healing, leaves a foreign body at the site of fracture, and does not offer any better functional results than does the conservative treatment.

An important point to be considered in the treatment and prognosis of fractures of the shaft of the long bones is the usually associated mechanical interference of the nearest joint, due to the changed long axis of the shaft; or to traumatism, resulting in arthritis. Reduction is here of prime importance, so that the joint-surfaces may be restored to the normal place; and early, but gentle, passive motion must be instituted, so as to prevent adhesions, at the same time, being careful not to increase the inflammation by traumatism from rough handling.

Fractures of the shaft of the clavicle usually result in some permanent deformity. The statement in the older text-books that fracture of the clavicle constitutes 19 per cent. of the fractures of the extremity is no longer true—at least, in urban populations. Hamilton states that the carrying of heavy loads on the shoulder and the recoil of overloaded guns are among the more common causes of fracture of the clavicle. As neither of these causes is present to any considerable extent in modern city life, it will be seen that accidents from these sources have been almost entirely eliminated. In 1000 cases of fracture of the upper extremity treated at the German Hospital, this fracture gives a total of but 2.7 per cent.

Formerly the most common site of fracture of the clavicle was the middle third; to-day, fracture of the acromial end is more frequently met with. (Fig. 3.) The cause of fracture of the acromial end of the clavicle is direct violence to the point of the

shoulder or indirect violence to the long axis of the humerus, driving the head of the bone against the acromion process. Impact of the elbow with the forearm at right angles to the arm, the line of force traveling along the humerus, results in fracture of either the acromial end of the clavicle or the acromion itself. In this locality there is practically no displacement, as the action of the conoid and trapezoid ligaments, from their oblique direction, serves to hold the fragments in apposition. The frequency of this injury was not suspected until demonstrated by the x-rays in a systematic search of all shoulder-injuries. The symptoms rather suggest "sprain" than fracture.

Fracture of the middle of the clavicle is always attended with considerable displacement, which is difficult and often impossible of reduction. Hence, union without deformity is rarely attained.

The inner fragment is pulled upward by the clavicular portion of the sterno-cleido-mastoid muscle, the strength of the pull being somewhat modified by the clavicular portion of the pectoralis major muscle. The outer fragment is pulled downward by the weight of the deformity and the action of the deltoid, at the same time being pulled inward by the action of the muscles arising from the trunk and attached to the outer fragment of the clavicle, to the scapula, and to the humerus; namely, the subclavius, the pectoralis minor, and the sternal portion of the pectoralis major and the latissimus dorsi. The outer fragment is made to rotate on its long axis by the action of the serratus magnus on the scapula. To correct the deformity, one must understand the antagonistic forces at work.

The indications are to carry the shoulder upward, backward, and outward, and so retain it. The position is secured by the Velpeau position, with a carefully adjusted axillary pad. The position can be maintained with the Velpeau dressing or with the modified Sayer's dressing of adhesive plaster. It is at times impossible to retain fixed dressings, on account of the patient's movements, particularly while asleep. The recumbent posture, with a low, hard pillow between the shoulders, gives the most satisfactory results; but patients rarely consent to the three weeks' confinement. In complicated fractures, such as multiple and comminuted fractures, and in fracture of both clavicles, the bed-treatment is strongly indicated. Operation should be attempted only when a jagged end of

FIG. 3.—Fracture of the acromial end of the clavicle.

FIG. 4.—Transverse fracture of the acromial process of the clavicle.

FIG. 5.—Fracture of the body of the scapula.

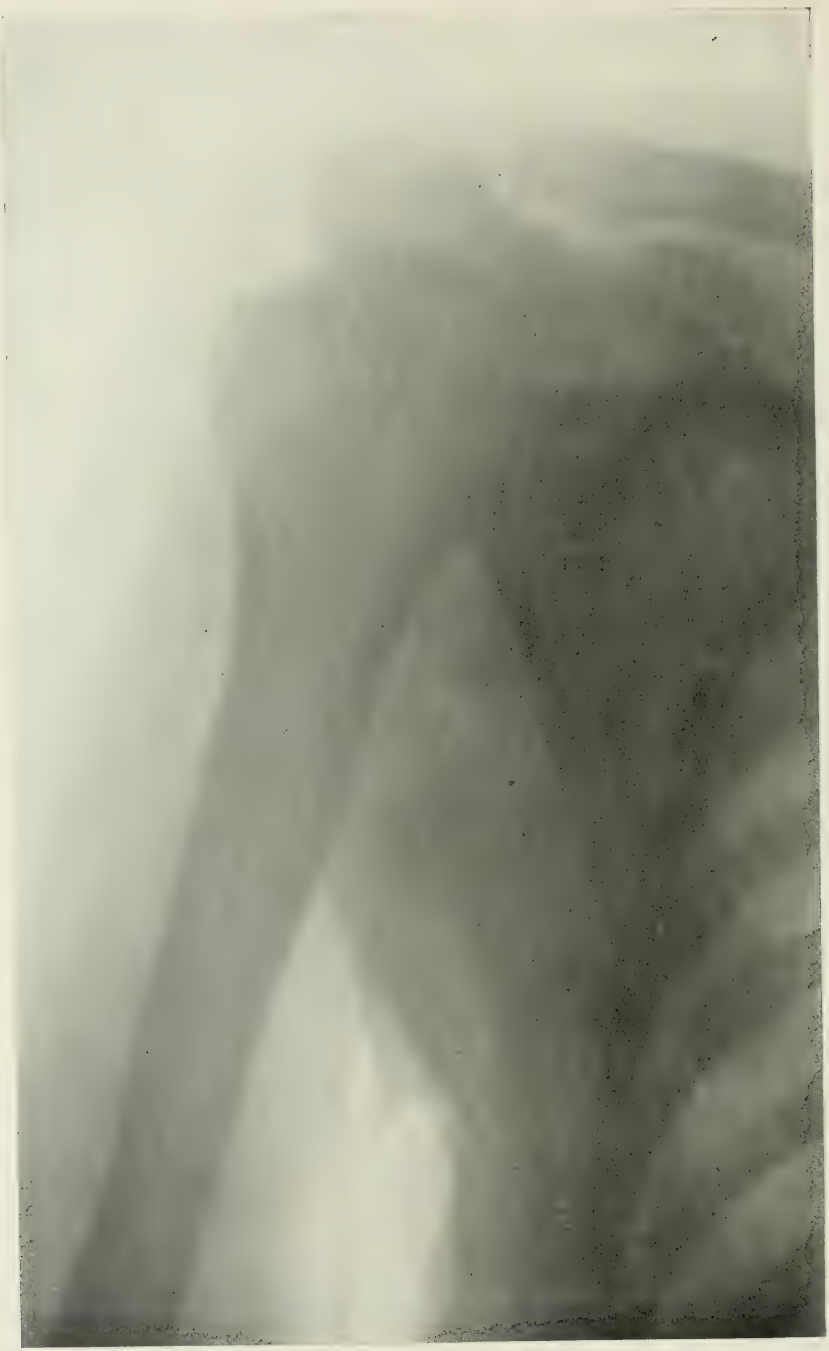


FIG. 8.—Fracture sprain of the head of the humerus.

bone threatens a neighboring vessel or nerve, or when the fragments are pressing on these structures so as to produce symptoms.

Fracture of the acromial process of the scapula (Fig. 4) occurs as a complication of luxation of the shoulder-joint or as the result of direct violence to the shoulder; and, indirectly, from the effect of a fall on the elbow, the line of force being along the shaft of the humerus and the breaking strain being exerted on the acromial process. The character of the fracture associated with luxation is that known as sprained fracture, a small spicule or plate of bone being torn loose, owing to the ligament's being stronger than the bone to which it is attached. When the fracture is gross, the fragment can be felt slightly below the normal position; the rotundity of the shoulder is slightly diminished. Swelling soon obliterates this sign.

The treatment consists in lifting the arm by pushing on the elbow, the forearm being flexed; thus relieving the pull of the deltoid muscle on the acromial fragment. The arm is then fixed to the body, the hand resting on the chest of the opposite side.

Fracture of the coracoid process of the scapula is very much less frequent than fracture of the acromial process. In a series of 2500 cases of fracture of the extremities, it occurred seven times; while in the same series, the acromial, either alone or associated with other fractures about the shoulder, occurred 46 times.

The injury is usually caused by a sharp blow on the point of the shoulder. The fragment is displaced downward by the action of the short head of the biceps, the coraco-brachialis, and the pectoralis minor muscles, to which it supplies attachments. The displacement is, however, governed by the coraco-acromial ligament. If this is intact, displacement does not occur.

The treatment consists in placing the arm in the position described for the treatment of fractures of the acromion, thus relaxing the pull of the opposing muscles.

Fractures of the neck and head of the humerus (Figs. 5, 6, 7, and 8) are comparatively common. The deformity in fractures of the head and neck is very prominent, the gross fractures somewhat resembling subglenoid dislocation; while those of minor degree of the head are frequently overlooked, owing to the absence of deformity. The upper fragment is pulled upward and inward by the muscles attached to the greater and lesser tuberosities of the humerus:

the subscapularis, to the lesser; the supra- and infra-spinatus and the teres minor, to the greater tuberosity. The lower fragment is pulled inward by the pectoralis major, latissimus dorsi, and teres major. The humerus is tilted outward by the deltoid, causing the upper extremity of the lower fragment to present beneath and in front of the coracoid process.

To overcome this deformity, the elbow should be grasped, and extension made steadily and persistently downward and outward, until the muscles relax, releasing the lower fragment. The arm is now abducted and flexed, so as to bring the lower fragment into contact with the upper. The extension being maintained, the entire arm is lowered and fixed by a roller bandage to the side of the patient's body, which has been protected with powder or a thin pad of gauze. Additional support is supplied by a shoulder-cap of binder's board, thin light metal, or moulded plaster of Paris.

The forearm should be supported at right angles to the arm with a narrow sling passing under the wrist. In this position, the weight of the forearm and of the arm below the seat of fracture is utilized for continuous traction. Additional traction can be applied by a weight, never over five pounds, attached to the arm below the fracture, on the principle of Buck's extension for fracture of the thigh-bone.

A piece of adhesive plaster (zinc oxide is less irritating to the skin) is cut two or three inches wide, and long enough to go from one side of the arm, an inch below the fracture, under the elbow, and up to a point opposite the starting point. The loop under the elbow should hang at least two inches below the olecranon process. A piece of board, the width of the strip and long enough to keep the plaster-strip from touching the condyle, is laid in the center of the strip and fixed with another piece of plaster extending above the lines of the condyle. Through a hole made in the center of the board comes the stout string for the support of the weight. The apparatus is adjusted by applying the adhesive strip to the inside and outside surfaces of the arm, reinforced with two or three narrow strips encircling the arm. Great care must be exercised in adjusting the circular strips, so that constriction of the circulation may not occur. A wise precaution is to have the strip a little short, the ends not meeting by an inch; so that if swelling does occur,

FIG. 9.—Fracture of the shaft of the humerus below the insertion of the deltoid.

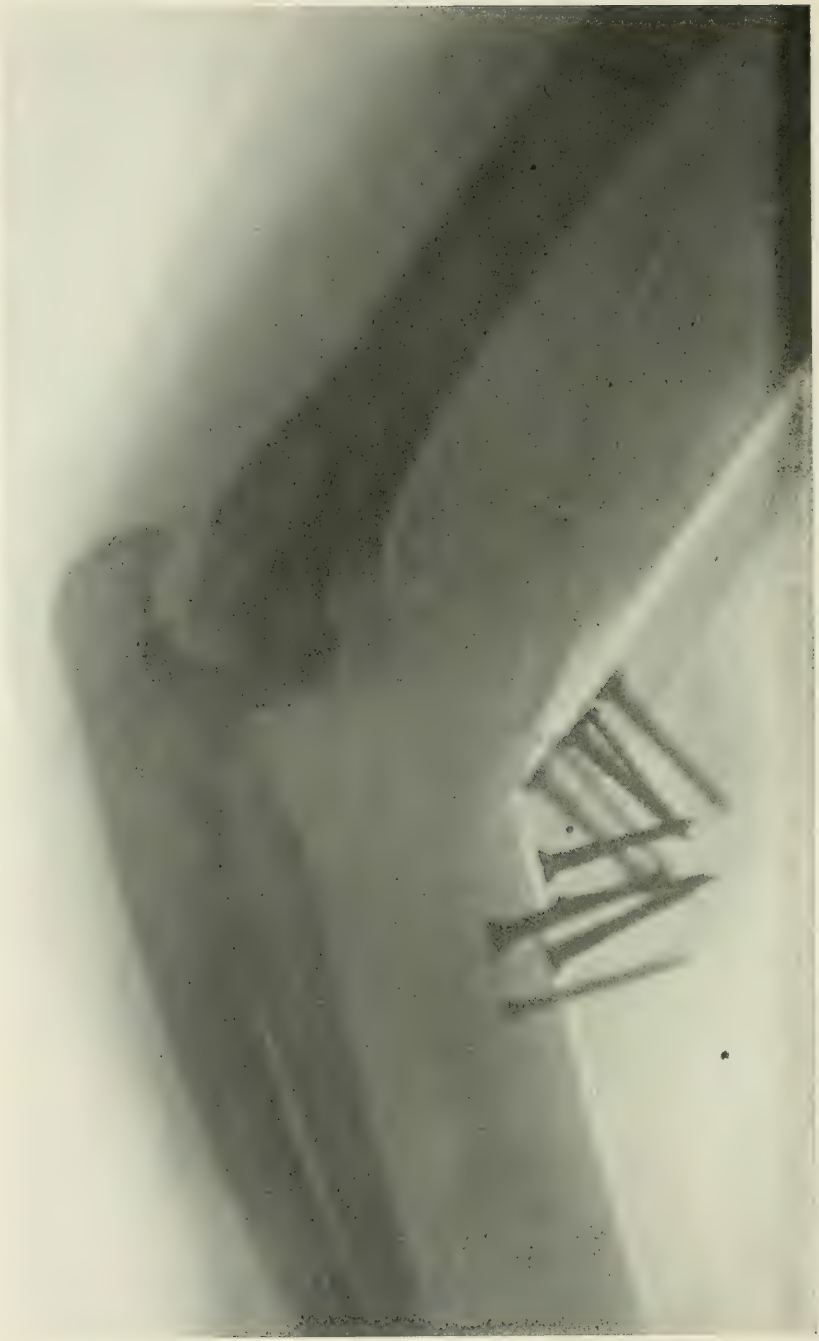


FIG 10.—Backward luxation of the ulna partially reduced.

FIG. 11.—Longitudinal fracture of the lower end of the humerus.

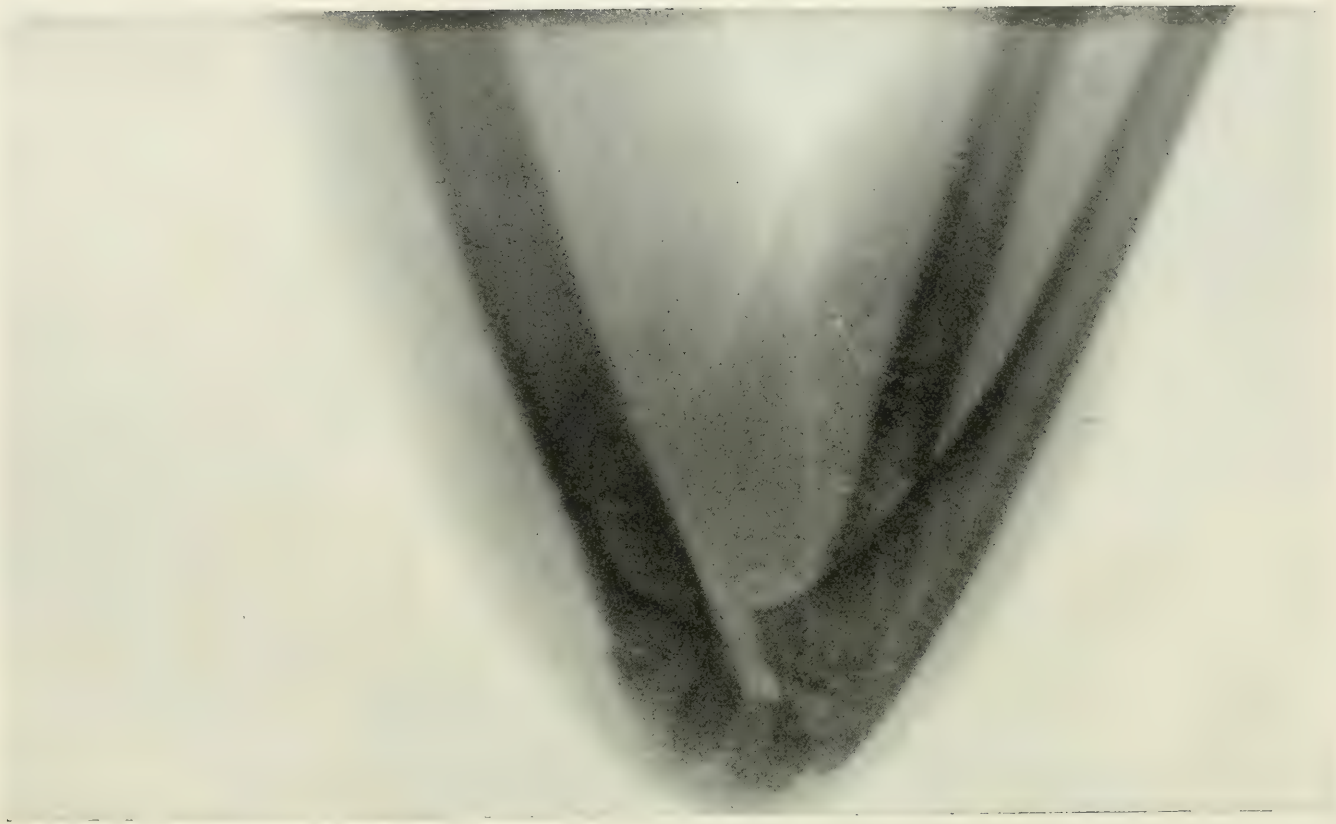


FIG. 12.—Fracture of the lower end of humerus, showing complete reduction in Jones's position, the coronoid acting as a fulcrum.

FIG. 13.—Fracture of lower end of humerus, showing faulty position due to incomplete reduction and improper angle of fixation.

FIG. 14.—Fracture of the lower end of the humerus. (Same as Fig. 15.)

FIG. 15.—Fracture of the lower end of the humerus, showing the action of the muscles concerned in the deformity. (Same as Fig. 14.)

FIG. 16.—Fracture of the lower end of the humerus. (Same as Fig. 17.)

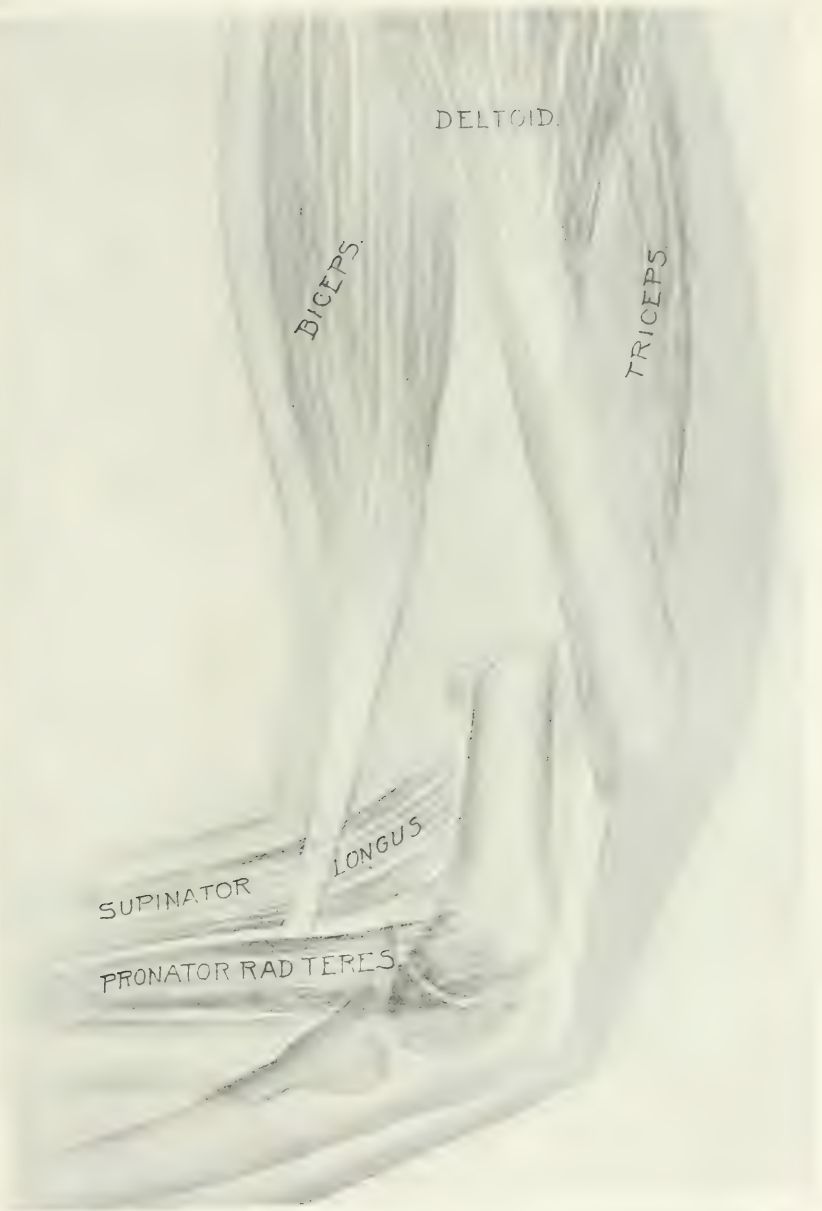


Fig. 17.—Fracture of the lower end of the humerus, showing the action of the muscles concerned in the deformity. (Same as Fig. 16.

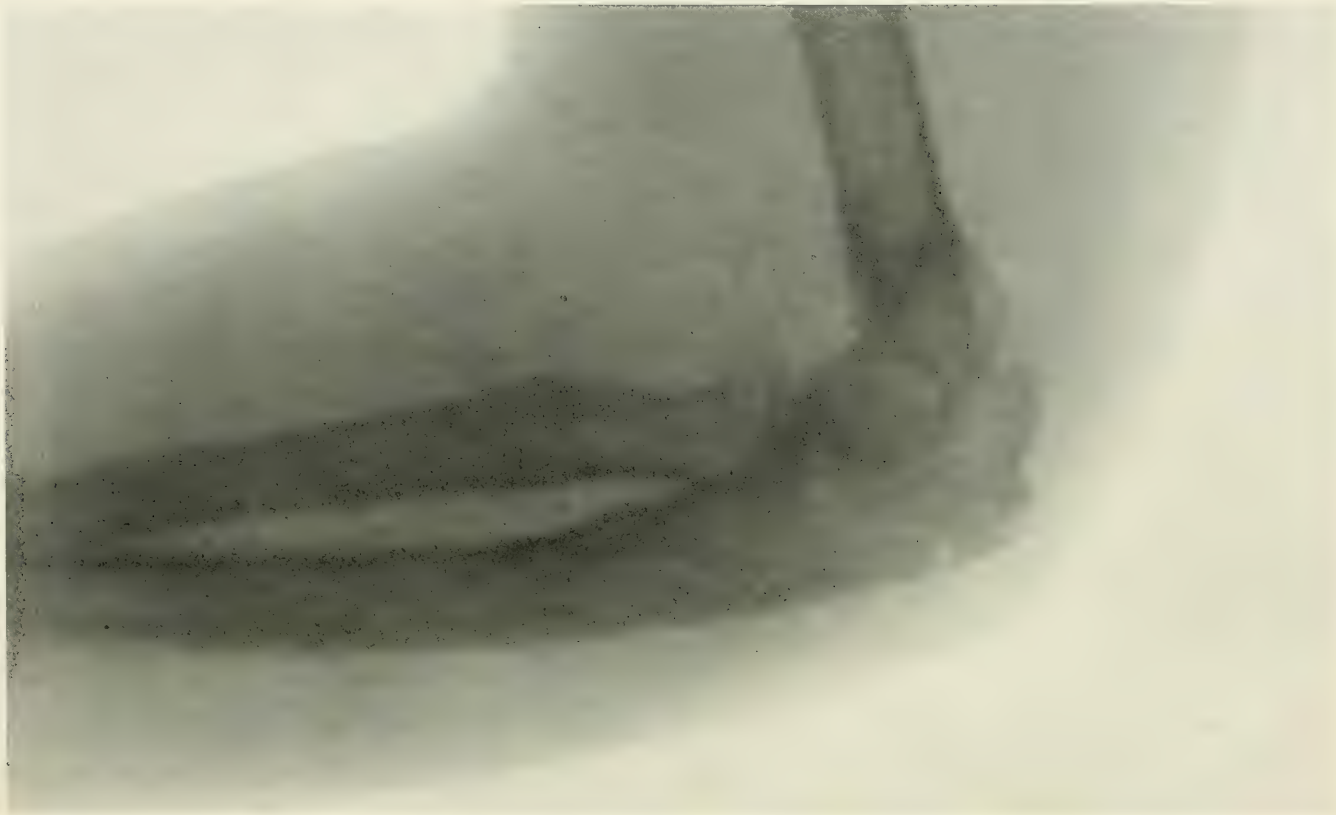


FIG. 18.—Comminuted fracture of the olecranon without displacement of the fragments

they will give to the increasing diameter. Two weeks is ordinarily long enough to accomplish the object in view.

Persistent deformity occurring within one, two, or three weeks of injury, due to insufficient traction, can frequently be overcome by the use of forced extension. It is well to apply the adhesive-plaster extension-strip to the arm before reduction is attempted, so that the adjustment of the fragments may not be jeopardized by the manipulations. The weight can be hung on when the rest of the dressing is completed. In fracture of the shaft below the middle, forced extension may be applied by hanging the weight over an internal-anterior angular splint used to fix the fragments.

If fixing the arm to the body with the use of forced extension fails to keep the fracture reduced, it will be necessary to dress the arm in abduction; thus bringing the lower fragment into contact with the upper, which is being held in its original position. This can best be accomplished by putting the patient to bed. The forearm is flexed at right angles and abducted; and traction is effected as already described, except that the weight must exert its pull over a pulley.

In fractures of the shaft of the humerus below the insertion of the deltoid (Fig. 9), the deformity depends largely on the line of fracture. When transverse, little or no deformity occurs; while in oblique fractures, a great deformity is noted, with considerable shortening of the limb. The continued action of the biceps and brachialis anticus, in front, and of the triceps behind, pulls the lower fragment upward, overriding the upper fragment either backward or forward, according to the direction of the line of fracture.

This fracture is best treated by using the body as the internal splint, with short humeral splints on the outside and in front; or, if near or below the middle of the shaft, an internal angular splint can be used instead of the body. Extension, which is so essential in all fractures of the humerus above the elbow-fractures, can be provided as already directed.

Oblique fracture of the humerus just above the condyle so closely simulates backward dislocation of the radius and ulna—and, in youth, separation of the epiphysis of the humerus—that we must be on our guard lest we make a serious error in diagnosis. (Figs. 10 to 17.) If the line of fracture is from above downward and for-

ward, the action of the biceps and brachialis anticus, in front, and of the triceps, behind, will draw the lower fragment upward; while the stronger action of the triceps pulls it backward. The diagnosis is made by noting the presence of crepitus, which is absent in luxation, and of increased mobility in the seat of fracture. The deformity in the fracture can be reduced by extension, but at once recurs when extension is discontinued; while luxation, once reduced, remains so. An anterior right-angle splint, well padded at the angle, extending up the arm on a level with the insertion of the deltoid, and below to the root of the fingers, is the proper splint for this fracture. Passive motion should be instituted at about the end of the second week.

Under fractures of the elbow we will class those occurring near the joint, as well as those directly involving the joint. They may be classified as follows:

Supracondyloid.—Internal and external condyle, and external condyle of the humerus.

T-fractures.—Longitudinal fractures of the lower end of the humerus (Fig. 11).

Head of the radius, olecranon process of the ulna, and coronoid process of the ulna.

Fracture of the internal condyle is important, because it destroys the carrying angle of the arm. The fragment is displaced upward and backward, carrying with it the ulna; and, in extension, the ulna projects backward. All other fractures of the humerus about the elbow should be carefully reduced by extension, making traction on the forearm, which is flexed to more than a right angle and in the position of supination. The most important point in the treatment of this class of fractures is complete reduction, anesthesia being employed, if necessary. The most useful dressing, and the one indicated in nearly all the fractures about the elbow, is the anterior right-angle splint made of wood. It should be well padded, and should extend from the root of the finger to a point about three or four inches below the shoulder. It is applied as follows:

The upper end of the splint is secured to the arm by two circular turns of adhesive plaster, one at the extreme upper end, and the other just above the angle. This second turn will prevent slipping when the forearm is flexed. The forearm is firmly grasped with one

hand, making extension, the other being used to manipulate the fragment into place. The forearm is slowly flexed to the position of the lower limit of the splint, extension being maintained, and fixed there with a turn of adhesive plaster just above the wrist. A roller-bandage is applied over all, and the extremity is supported with a narrow sling at the wrist.

Fracture of both condyles, T-fracture, oblique fracture of lower end of humerus, and fracture of the coronoid process of the ulna form the exceptions to the above-described method, and are best treated in the position of extreme flexion—Jones's position. (Figs. 12 and 13.) Some authorities recommend for this class of injuries continuous traction with the arm at full extension, the patient being confined to bed for the purpose.

The position of acute flexion must be used with judgment, care, and constant watchfulness, so as to prevent and to correct promptly pressure-symptoms on the blood-vessels. It is of the first importance to reduce the fracture—as the position in the absence of reduction is painful, will give bad results, and is dangerous. The dressing is applied as follows:

The fracture is completely reduced, anesthesia being employed, if necessary; for this treatment, as well as all others, fails, if reduction is incomplete.[1] The humeral condyles are grasped between the finger and thumb of one hand, while a finger of the other is placed in the bend of the elbow. Traction is made upon the forearm, and it is slowly flexed to an acute angle. While the forearm is being flexed, traction and external pressure are brought to bear upon the loose fragment of the humerus, to correct the deformity. These manipulations will materially assist in the reduction.[2]

The degree of flexion must be determined by the obstruction offered by the local swelling. If the swelling is great or is likely to increase very much, the degree of flexion must be less than when there is no swelling. In the bend of the elbow, to prevent chafing, is placed a piece of gauze upon which has been dusted dry powder.

The position of extreme flexion is maintained with a handkerchief-bandage encircling the wrist and tied around the patient's neck, the entire arm and forearm being fastened to the body with a

[1] Scudder, Treatment of Fractures, p. 197.

[2] Scudder, p. 199.

roller-bandage or a strip of adhesive plaster. In persons of intelligence who have reached the age of discretion, the neck-sling will be sufficient; for so long as the angle of acute flexion is maintained, there is little danger of displacing the fragments.

The radial pulse is the safety-valve, and should be felt at frequent intervals during the first few days of the dressing. The swelling is also an indication of trouble; for if it increases, the angle of flexion must be made less acute. This dressing, while not overly comfortable, should not be painful—following the rule that if a fracture-dressing is painful, it is incorrectly applied, or the fracture is wholly or partially unreduced.

Fracture of the olecranon process of the ulna is not always productive of the wide separation erroneously mentioned in the classical descriptions of this fracture. (Fig. 18) Comminuted fracture of this process and simple fracture due to direct violence occur without upward displacement of the upper fragment, owing to the fact that the periosteum and the ligamentous structures attached to it are not torn and hold the fragments in contact. If displacement is present, it is due to the pull of the triceps muscle.

The fracture should be treated with the arm in full extension on a straight, well-padded board-splint, extending from the level of the insertion of the deltoid to the root of the fingers. Strips of adhesive plaster, looping over the upper fragment and brought around to the front of the forearm, will aid in reducing the fracture; although, in the most favorable circumstances, fibrous union is the usual result.

Fracture of the head of the radius occurred 27 times in the series of 2500 cases already quoted. It is, therefore, common enough to warrant a careful search in injuries about the elbow. The diagnosis is rather difficult to arrive at from clinical evidence alone, as the thick mass of muscles covering the location of the fracture renders the slight deformity obscure. The movements of pronation and supination are lost; but, if examined before swelling is present, crepitus can sometimes be elicited by rotating the forearm with one hand, the thumb and fingers of the other grasping the head of the bone. The upper fragment is drawn slightly upward by the biceps, and inward by the pronator radii teres. The degree of displacement depends upon the integrity of the orbicular ligament.

FIG. 19.—Fracture of the ulna, showing ring callus, but no deformity.

FIG. 20.—Fracture of the ulna. (Same as Fig. 21.)

FIG. 21.—Fracture of the ulna, showing the action of the muscles concerned in the displacement. (Same as Fig. 20.)

FIG. 22.—Fracture of both bones of the forearm.

FIG. 23.—Side view of the fractures shown in Fig 22.

The treatment consists in flexing the forearm to a right angle with the arm, the forearm being retained in the position midway between pronation and supination. An internal right-angle splint meets the indications.

Fracture of the head of the radius within the confines of the pronator radii teres gives rise to very marked displacement of the fragments. The upper fragment is strongly supinated by the biceps and the supinator brevis, while the biceps also flexes and draws the fragment forward. The lower fragment is pronated and drawn toward the ulna by the pronator radii teres and the pronator quadratus. By flexing the forearm in supination, the lower fragment is brought forward; thus meeting the upper fragment, which cannot be manipulated. In any other position, the fracture would heal with the lower fragment fast to the ulna and without bony union, causing loss of the movements of pronation and supination. An anterior right-angle splint and a short posterior splint to the wrist will furnish the necessary fixation.

In fracture of the radius below the insertion of the pronator radii teres, the upper fragment is held midway between pronation and supination, and is pulled upward by the biceps. The lower fragment is held in pronation by the action of the pronator quadratus, being also pulled toward the ulna by the same muscle. The pull of the supinator longus on the styloid process, into which it is inserted, will throw the upper end of the lower fragment toward the ulna. To overcome the displacement, the forearm should be flexed on the arm, to relax the pull of the biceps. Dressing the arm midway between pronation and supination on an internal right-angle splint will bring the lower fragment into apposition with the upper. The hand should not be fixed on the splint, but should be allowed to fall toward the ulnar side; so that its weight may make continuous traction on the lower fragment, tilting its upper end away from the ulna.

In fracture of the shaft of the ulna, the upper fragment remains undisturbed; while the lower is pulled toward the radius by the pronator radii teres. This fracture should be dressed midway between pronation and supination, the anterior splint extending to the root of the fingers, so as to do away with the pull of the unsupported hand. (Figs. 19, 20, and 21.)

In fracture of both bones of the forearm, (Figs. 22, 23, 24, 25, and 26) the lower fragments are drawn upward by the combined actions of the flexor and extensor muscles. The lower fragments will override the upper, either anteriorly or posteriorly, according to the line of fracture. The pronator quadratus will pull the lower fragments toward each other, the radius being in a state of pronation. The action of the biceps and pronator radii teres pulls the upper fragment upward and forward. The brachialis anticus draws the upper fragment of the ulna slightly forward, but not on the same level with the radius. The displacement can be overcome by extension at the wrist and counterextension at the elbow, the forearm being flexed. The arm should be dressed on an internal right-angle splint, reaching to the tips of the fingers; and a short posterior splint, to the carpus.

In case of persistent deformity, a narrow splint placed over the position of the interosseous membrane will help to overcome the action of the pronator quadratus. A lead-pencil or tubular rubber will meet the indication. Such a dressing must be carefully watched; as too great pressure, or pressure too long continued, will produce pressure-necrosis and ulceration.

Fractures of both bones of the forearm should be treated with an anterior and posterior board-splint. The anterior support is best applied by using the internal right-angle splint, having a hole cut in it for the accommodation of the internal condyle of the humerus. It should extend from the axilla to the base of the finger. It thus immobilizes the elbow and wrist joints, the joints at each side of the fracture. The splint places the forearm midway between pronation and supination, with the thumb up. In this position, the radius and ulna are most widely separated; and the patient is most comfortable, as the opposing muscles are at rest. It is often extremely difficult to reduce fractures of both bones of the forearm; and sometimes complete reduction is impossible, short of operation. However, the attempt should be made under anesthesia, the dressing being applied before the patient comes out of the anesthetic. In case of failure, operation and reduction may be done more safely and as efficaciously at the end of the third or fourth week, as later.

Massage and early passive motion, persevered in, will frequently work wonders in the injury, restoring almost normal function, even if there is some persistent deformity. I doubt whether reduction

FIG. 21.—Fracture of both bones of the forearm.

FIG. 25.—Fracture of both bones of the forearm. (Same as Fig. 26.)

FIG. 26.—Fracture of both bones of the forearm, showing the action of the muscles concerned in the displacement. (Same as Fig. 25.)

in both bones of the forearm is ever complete, short of operation; yet restoration of function with a minimum of deformity is the almost invariable result.

Fracture of the lower end of the radius, the most common fracture occurring in the adult, is one of the most important injuries we have to deal with. It jeopardizes the motions of pronation and supination, flexion and extension, and adduction and abduction of the hand, the prehensile motion of the thumb, and extension and flexion of the fingers. Its exposed position makes appearance an important consideration. It therefore behooves the surgeon to apply all his skill in treating this fracture. Reduction is requisite for success; for, when successfully reduced, it is readily retained. At a meeting of the College of Physicians of Philadelphia, December 2, 1851, Dr. Henry Bond read a paper on Fractures of the Lower End of the Radius and their Management. In it he described a splint and a mode of dressing that so exactly meet the indications of this class of fractures that the splint has ever since borne its originator's name and has retained its popularity—at least, in Philadelphia.

The principle upon which the dressing is founded is the fact that the hand is at rest midway between pronation and supination, with the fingers partially flexed, the end of the thumb and the index finger being nearly in apposition (pen-holding position). The muscles of extension and flexion being relaxed, a minimum amount of tension results. Dr. Bond pointed out the following objections to the anterior and posterior splint dressing:

"In the first place, it violates what ought to be regarded as a surgical canon in the treatment of fractures, viz.: to adopt such a position as will put all the muscles acting on a part as much in repose, as free from tension, as possible; so that the least counteracting force will be required. Secondly, the constrained position of the hand demands tighter bandaging, in order to prevent derangement of the fragments by paralyzing or subduing the muscles that are rendered tired by the position assumed. Thirdly, this constrained position and tight bandaging greatly increase the danger of that protracted or permanent rigidity of the hand and fingers which is a too frequent result of these injuries. Fourthly, this mode of dressing—by long, straight splints—not only increases the

danger that it will result in rigidity, but that when it does occur, the hand will be left unsightly, inconvenient, or useless. Fifthly, there is another objection to it, which will be regarded by the surgeon as of more or less importance, according as he is actuated more or less by the feelings of humanity. I refer to the distress or comfort which must result from a constrained position and the force applied to maintain it."

"The muscles that act on the hand are least tense or most in repose when the hand is inclined backward, so that the metacarpus forms a considerable angle with the forearm, when it is also inclined toward the ulnar side of the arm, and when the fingers are moderately flexed. In this case, it will be perceived that the longitudinal axis of the forearm, if prolonged, would not correspond with that of the hand, but would pass through or very near the point where the thumb and index finger most easily and naturally meet. . . . This will be found to be the position of the hand when it hangs by the side with all the muscles relaxed."

With the foregoing facts in mind, it will be seen that Bond's splint meets all indications, as he himself says:

"(1) It maintains such an inclination of the hand upon the forearm as shall most effectually relieve the muscles from tension or put them at rest.

"(2) It maintains the hand and fingers in a position that, if rigidity should result, would make the member as little of an encumbrance, and would retain as many of its uses, as possible.

"(3) It is easy of application, requiring no extraordinary skill or dexterity, and is little liable to become disarranged or displaced.

"(4) It makes the dressing easy and comfortable, while it does not lack efficiency."

Early massage and passive motion are strongly indicated in fractures about the wrist, and should be instituted at the end of the first week. Four dressings are all that are ordinarily required, one week apart. The injured member is gently, skilfully, but thoroughly rubbed and moved at each dressing; and this rubbing and moving is continued every two or three days for several weeks after the dressings have been discarded.

Reduction must be complete, if a happy result is to be secured. The method of reduction is as follows: Grasp the hand of the

FIG. 27.—Fracture of the scaphoid bone.

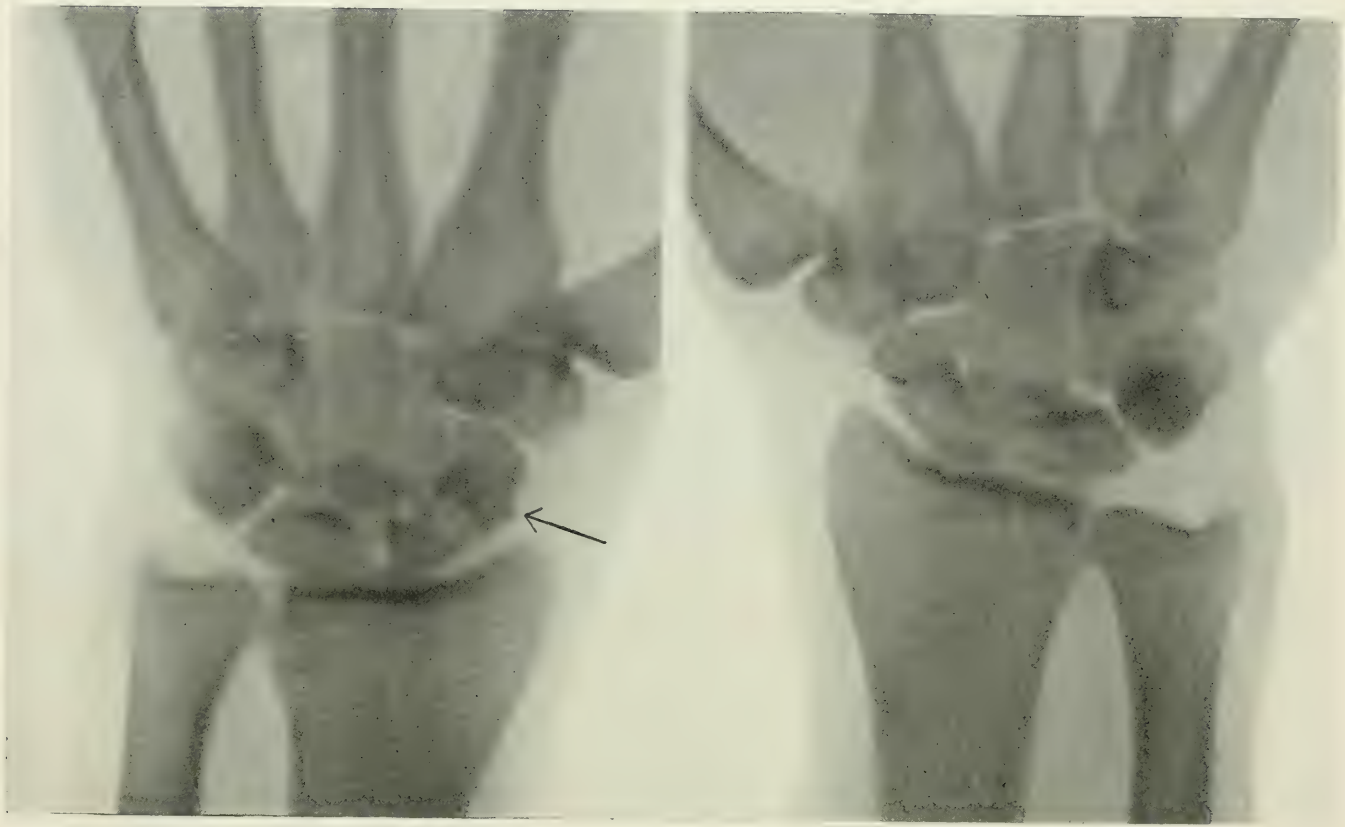


FIG. 28.—Comminuted fracture of the scaphoid of the left hand. (The right hand for comparison.)

injured limb as you would in shaking hands, right hand with right hand, or vice versa. Make strong, even extension and strong abduction. The thumb of the other hand makes pressure on the dorsal side of the forearm, while the finger on the palmar surface makes counter-pressure, moulding and pushing the fragments into position. Without relaxing the extension or the pressure on the now reduced fragments, a Bond's splint, padded with wadding, and having a palmar pad placed so that it will make pressure on the upper fragment, is slipped under the forearm, holding the fragment against the splint. The fingers are now placed on the palmar pad, which the patient is instructed to grasp. A dorsal pad over the lower fragment is placed in position, the whole being retained with a roller-bandage. It is well to have the patient extend and flex the fingers during the entire splint period of treatment, this action being rendered possible by the use of this splint.

In the reduction of uncomplicated fractures of the wrist, the range of motion and usefulness should be nearly normal at the end of four weeeks, or when the splint is discarded. In some cases of impacted fracture of the lower end of the radius, when the deformity is not great, it is wise to make no attempt at reduction. Inform the patient of the facts, and persist in the passive motion and massage, the result being restoration of function with a minimum of deformity.

Fractures of the carpal bones are far more frequent than was formerly thought, the scaphoid (Figs. 27 and 28) and semilunar being most often fractured, and occasionally the os magnum. In the series of 2500 cases already referred to, fracture of the carpal bones occurred 146 times. There were 87 uncomplicated, and 59 associated with fracture of neighboring bones as follows:

Carpus and radius	36
Carpus and ulna	7
Carpus, ulna and radius	12
Carpus and metacarpus	3
Carpus, metacarpus and phalanges	1

The history of a fall on the hand, especially when the hand is in full extension or flexion, with a following sharply-localized area of tenderness and of circumscribed swelling and pain over the carpal bones, is sufficient to make a diagnosis. The x-rays will give invalu-

able corroborative information. Such fractures should be treated in a fixed dressing, and I know of no better dressing than Bond's splint. The pathology of this fracture illustrates the pathology of that rather vague injury to the joint known as sprained wrist.

It is not always wise to reduce forcibly an impacted fracture. Healing is sometimes more satisfactory in unmolested impaction than when reduced. This is particularly true in the very common and distressing fractures in the aged—fractures involving the hip-joint. In cases of impaction at the wrist, reduction had better not be attempted unless the deformity is very great. Impacted fracture of the anatomical neck of the humerus is not a very common accident in the adult, especially in advanced years. It is frequently diagnosed bruised or sprained shoulder, owing to the lack of displacement; the neck of the bone being thicker than normal, crepitus is absent. It should not be reduced, but should be dressed in the same way as fracture of the surgical neck, with the difference that passive motion should be started early (at the end of the first week), and that the fixed dressing should be applied not longer than three weeks, so as to secure early active motion.

The rule of procedure in treating compound fractures is to operate formally on these cases as soon as the period of shock has passed. In the meantime, the wound should be protected by a wet bichloride towel, and the limb should be elevated.

The patient, having been etherized, the injured limb should be shaved and thoroughly cleansed with soap, hot water, a brush, alcohol, and bichloride-solution, the nails being pared and thoroughly cleansed. If the line of fracture is single and the wound small, thorough sponging with a weak antiseptic solution composed of mercury bichloride (1 to 2000) and carbolic acid (1 to 30) is sufficient. The fracture should now be reduced, and an aseptic gauze-dressing should be applied. Over this, a plaster of Paris cast is adjusted, the fragments being held in apposition by an assistant, making extension on the distal portion of the limb. A plaster cast on such a fracture should be cut before it hardens, so as to allow for swelling. If the wound be large or the fracture comminuted, the wound should be generously enlarged and thoroughly cleansed of all blood-clots and shreds of fascia or muscle. Such a wound permits of the removal of loose fragments of bone and

the proper fixing of the main fragments by wiring, plating, or pegging. The wound of a compound fracture should never be sewed up tight. Drainage is essential, and an open wound is usually sufficient. In case of extreme laceration and free bleeding, rubber-tube drainage is necessary, but should not traverse the diameter of the limb. It should lead from the seat of fracture to the exterior, a counter-opening being made, if necessary, opposite the original skin-wound. It can be retained in place with a stitch through the skin and the tube.

EXPERIMENTAL RESEARCHES BEARING ON SURGICAL INTERVENTION IN NEPHRITIS[1]

A LECTURE DELIVERED IN THE DEPARTMENT OF CLINICAL MEDICINE AND PATHOLOGY OF THE ROYAL UNIVERSITY OF BOLOGNA, ITALY

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GENTLEMEN: Five or six years ago the question whether or not we should resort to surgical intervention in acute and chronic nephritis not amenable to medical measures, was not even considered; to-day it is discussed everywhere, but cannot yet be considered definitely answered. We are dealing with a therapeutic problem the solution of which would be a great boon to the world, since, as is well known, in spite of our most careful and rational medical treatment, we often see diffuse nephritic processes progress toward a fatal termination and are absolutely powerless to prevent this. Knowing that the disease always progresses toward a fatal issue, the query, in accordance with a well-known aphorism, whether it is not better to experiment with means of uncertain or at least doubtful value than to remain inactive in the face of death, is bound to arise. Should not the attempt be made to save by means of an operation, that is, by means of decortication of the kidney, the lives of those who are in danger? The literature on the subject of this operation, which was suggested but a few years ago, has grown to such a volume that it would lead me astray were I to attempt to summarize it.

Chance or errors in diagnosis have repeatedly led to the discovery of important and useful operations, and it was an error in diagnosis which, less than 10 years ago, led to the discovery of the value of decortication of the kidney in nephritis,—just as in 1863 the fortunate error of Spencer Wells taught him the usefulness of laparotomy in the treatment of tuberculous peritonitis, and the error of Drummond and Morrison brought forth in the brilliant

[1] Reported and translated by Leon Lebovici, M.D. (Vienna), Carlsbad, Bohemia.

mind of Talma the idea of fixing the omentum to the abdominal wall in order to establish a collateral circulation in cases of hepatic cirrhosis. Thus, it was in 1896, that Harrison, a distinguished English surgeon, having erroneously diagnosed a case as one of renal calculus, practiced decortication of the kidney and found the organ the seat of a nephritic process only; the patient's complete recovery led Harrison to perform the same operation on other patients similarly affected, and he obtained good results. The benefits thus derived from decapsulation of the kidney in renal congestions and inflammations was likened by Harrison to those obtained by iridectomy in glaucoma, by incision of the tunica albuginea in orchitis, or of the skin and subcutaneous tissue in phlegmons; that is, the operation diminishes the reno-capsular tension caused by the inflammatory and hemorrhagic processes in the kidney. Harrison's method, as it is known, was taken up by Israel in Germany, by Mossou, Tuffier, Jaboulay, and Talamon in France, by Rovsing in Denmark, by Elliot in Chicago, by Giordano in Venice. Taking full advantage of the numerous opportunities for scientific research at his disposal in the hospitals of New York, Edebohls, in one of his recent publications gives statistics of about 60 cases of nephritis operated on by this method with the most excellent results. He reports 18 per cent. of permanent recoveries, and 40 per cent. of marked improvements. These results were recently confirmed by Ferguson of Chicago, who expresses the hope that when the indications for surgical intervention are better understood, the operative mortality will be reduced to 5 per cent. On the other hand, the value of these statistics was attacked in 1902 by Senator, and more recently by Rosenstein, both of Berlin, by Kümmel and Riedel in Germany, and by Ferranini in Italy; they claim, that not all of Edebohl's patients had nephritis, but that a large number were suffering from nephroptosis and congestive renal disturbance—which are always benefited by decortication. A summing up, however, of the surgeons for and against operation in the treatment of nephritis, permits me to arrive at the conclusion that from 70 to 80 per cent. of those having tried the method, speak of it in terms of confidence and enthusiasm.

After this brief summary of the history of the operation, I wish to point out to you the results of some experiments, which I have

performed to determine the influence of renal decortication in acute nephritis.

Method of Research.—I purposed to solve the important clinical problem, whether scarification or decortication of the kidneys could arrest, improve, or modify the nephritic process experimentally induced in rabbits by hypodermic injections of cantharides or diphtheritic toxin. I also tried to induce chronic nephritis by means of lead salts and chromic acid; but in this I failed and I am, therefore, only able to report to you on acute processes similar to the acute intoxications or infections of man. Each experiment was carried out as follows:

Two rabbits of the same age and weight were injected with an equal dose of a solution of cantharides or diphtheria toxin, and after signs of nephritis became manifest (albumin, tube casts, red and white blood corpuscles), I operated on one of the animals and kept the other for control. Scarification of the capsule being found less efficacious than decortication, I performed decortication of one kidney in nearly all instances; occasionally I operated on both kidneys, with a few days intermission. But as the animals bear the double operation badly, it is preferable to limit the procedure to unilateral decortication.

The operation in young rabbits is very easy; and as I always observed the strictest antisepsis, I never had any postoperative infection. To get the best results, however, it is necessary to use robust rabbits, from 4 to 5 months old, and weighing from 1.5 to 2 kilograms, because younger and smaller animals, when operated on during the colder months, frequently do not recover from the operation. Before and after decortication the animals were kept in special cages, having a metallic receptacle in their floor for collecting the urine; this was measured and examined daily, and sometimes twice or thrice a day.

An exact account of the variations in weight was kept of all the rabbits, and when they died or were killed an autopsy was performed; the kidneys were fixed in Flemming's solution, in alcohol, and in potassium bichromate. Some of the rabbits were killed soon after the albumin disappeared from the urine; others were killed in from 15 to 35 days after the nephritis had subsided. To note the effect of decortication on the healthy kidney I operated on 2

animals, a rabbit and a dog, killing them 25 and 30 days, respectively, after the operation.

As had other experimenters, I also found the operated kidney to be attached to the peritoneum by adhesions containing delicate blood vessels; the newly formed renal capsule was thin and could easily be detached from the subjacent tissue. I was also able to demonstrate, agreeing in this with Ferranini, that the newly formed blood vessels, arising from the neighboring organs and running into the kidney through these adhesions, completed anastomoses with the stellate and straight veins of the parenchyma, thus increasing the blood supply of the renal glomeruli and tubules.

Researches with Cantharides.—Eight rabbits were injected with from 3 to 4 milligrams of cantharides each. In every case after the second or third injection a diminution of the urine associated with albumin, hyalin and bloodcasts, red and white blood corpuscles occurred. In 2 of the rabbits I scarified the kidney, and saw the albumin disappear from the urine of both within a few days. In 2 others I performed unilateral decortication; these animals also recovered within a short time.

Of the 4 control rabbits 2 recovered spontaneously, presenting neither albumin nor blood in the urine 15 and 20 days respectively from the date of the cantharides injection. The other 2 rabbits died after 12 and 17 days respectively; at the autopsy I found severe, diffuse lesions of the glomeruli and tubules of both kidneys.

In the operated rabbits which recovered and were killed at intervals of from 15 to 35 days after operation, the inflammatory process had disappeared in part or altogether.

Researches with Diphtheritic Toxin.—Twenty rabbits were injected with moderate doses of diphtheritic toxin to prevent a rapid death from intoxication. Ten of the rabbits, the control animals, died in from two to four days after the injection; all presented the well known symptoms of acute nephritis. At the necropsy the kidneys were found enlarged, of diminished consistency, congested, and covered with small hemorrhages. On section the cortex and medulla were found distinct, the cortical substance increased in thickness, pale and somewhat yellowish in color; the medullary substance was congested. The microscopic examination showed congestion and hemorrhage of the Malpighian bodies; Bow-

man's capsules were distended, filled in some places with blood, in others with an amorphous homogeneous substance; in some instances this was finely granular; the epithelium lining the glomeruli and the renal capsule was either partly or wholly destroyed. The epithelium of the convoluted tubules was swollen; at some points the cells had fused, at others they were disintegrated; at other points there were evident signs of coagulation necrosis. There was no trace of fatty degeneration. The epithelium of the straight tubules and of the loops of Henle was in a better condition, but the cells were distended, their protoplasm transparent and full of vacuoles. Extensive hemorrhage from the capillaries and intertubular vessels was present. The uriniferous tubules of the cortex and the medulla contained many hyalin and a few blood and epithelial casts.

The remaining 10 rabbits were operated on by different methods. In one rabbit I scarified both kidneys in numerous places a day after the onset of albuminuria. The rabbit succumbed 12 hours after the operation; the autopsy revealed signs of very acute nephritis. The other nine rabbits had one kidney decorticated two or three days after the appearance of albumin and casts. Two animals succumbed one day after the operation, one six days, and one twelve days after. In the last-mentioned case no albumin had been in the urine for four days. The kidneys were on the road to recovery, but the animal had lost 400 grams in weight. The other four rabbits survived the operation and were killed respectively, 3, 14, 30, and 35 days after the operation. In them the following phenomena were observed: (*a*) a marked increase in the amount of urine secreted; (*b*) a disappearance of the albumin, the casts, the red corpuscles, leukocytes, and renal cells in from six to eight days after decortication; and (*c*) the macroscopic and microscopic examination revealed in progress the process of reproduction of the capsule of the operated kidney, together with the new formation of blood vessels, and the progressive resolution of the inflammatory process; this was not very marked six days after decortication, but was complete in from 30 to 35 days.

One rabbit, having traces of albumin in the urine 15 days after the decortication of the left kidney, had the other kidney operated on. The albumin then disappeared completely in from three to

four days, and when 35 days after the last operation and about two months after the first the animal was killed, the kidneys were found to have completely recovered; moreover during life the animal had increased in weight.

These results are also in accord with those obtained after similar experiments by Dr. Fabris, of the surgical clinic in Bologna.

The results may be tabulated as follows:

	OPERATION	RESULT	
Nephritis from Cantharides	Scarification	Recovered	2
		Died	0
	Unilateral Decortication	Recovered	2
		Died	2
	Control Rabbits	Recovered	2
		Died	2
Nephritis from Diphtheria Toxin	Bilateral Scarification	Recovered	0
		Died	1
	Unilateral Decortication	Recovered	4
		Died	1
	Bilateral Decortication	Recovered	1
		Died	0
	Control Rabbits	Recovered	0
		Died	10

Finally I may state the following facts:

(1) One month after decortication of one kidney a reproduction of the fibrous capsule is noted, together with a formation of connective tissue adhesions containing blood vessels, which go to increase the blood supply of the operated kidney.

(2) All the rabbits rendered nephritic by cantharides and subsequently operated on by scarifications or unilateral decortication recovered completely, whereas the death-rate of the control animals was 50 per cent.

(3) While bilateral scarification of the kidneys in nephritis due to diphtheria toxin did not prove efficacious, decortication, both unilateral and bilateral, saved the lives of 50 per cent. of the affected animals. The mortality among the control animals was 100 per cent.

(4) The effects of decortication are: Increase in the amount of urine, gradual disappearance of albumin and renal elements in the

urine, and resolution of the nephritic process in from 30 to 35 days after operation, a fact verified by the histologic examination of the organs.

(5) Decortication of one nephritic kidney is, by virtue of the well-known reno-renal reflex, also attended by a beneficial influence on the other diseased kidney.

Critical Consideration and Interpretation of the Observed Facts.—The question arises, By what mechanism does renal decortication exert its favorable influence on the course of acute nephritis? If it were proved that an alternation in the internal secretion of the kidneys and the production of the so-called nephrolysins really plays an important or exclusive part in the pathogenesis of nephritis, we might suppose that decortication has the effect of clearing the kidneys of many of these products, which injure them by their accumulation. But in spite of the valuable and careful researches on the internal renal secretion reported from many places, I cannot take it for granted that this alteration exerts a great or exclusive influence on the production of the nephritic symptoms. I admit that the cell products could act as local and general poisons, but I am of the opinion that the nephritis and its symptoms of general intoxication are chiefly due to poisons circulating in the blood and derived from chemical changes within the tissues and from organic metabolism (endogenous and exogenous poisons, bacterial toxins, leukomaines), as well as from toxic products derived from gastric and intestinal putrefaction, etc. I am supported in this view by the very thorough work performed on chronic nephritis by Severino Tino, the results of which are in opposition to the theory maintained by Ascoli, according to which the nephritic symptoms depend upon the production of nephrolysins and the altered internal secretion of the kidneys. Tino made a careful study of 43 cases of chronic parenchymatous nephritis and 39 cases of interstitial nephritis (primary and secondary), observed in the medical clinic at Bologna, and reports a close relation to exist between urinary retention and the appearance of urinary phenomena.

The cases referred to were divided into four classes, depending upon the duration of the disease and the length of time of observation at the clinic, where careful analyses of the urine were made daily or at short intervals.

Cases of Nephritis.		Average quantity of urine in 24 hours, in c. c.	Average quantity of solids in the urine in 24 hours, in grams.	Average quantity of urea in 24 hours, in grams.	Differences in the cases fatal from uremia, as to the average quantity of		
					urine, in c. c.	solids, in grams.	urea, in grams.
First class. (Duration of less than 1 year).	Died of uremia	967	43.18	9.57	543	29.25	13.08
	Did not die . .	1510	67.43	22.65			
Second class. (Duration from 1 to 2 years).	Died of uremia	1074	35.96	18.79	348	14.01	4.81
	Did not die . .	1422	49.97	23.60			
Third class. (Duration from 3 to 4 years.	Died of uremia	914	34.70	11.24	17	19.34	
	Did not die . .	931	54.04				
Fourth class.	Died of uremia	810	36.17	10.20	1019	25.08	13.39
	Did not die . .	1829	61.25	23.59			
Secondary interstitial nephritis.	Died of uremia	1980	30.90	18.01	225	3.52	3.81
	Did not die . .	2205	34.42	21.82			
Primary interstitial nephritis.	Died of uremia	1412	28.24	15.95	764	15.28	16.82
	Did not die . .	2176	43.52	32.77			

This table demonstrates three facts which are in accord with the theory attributing the uremic phenomena to urinary retention or renal insufficiency, and which could not be explained by the nephrolysin theory; they are as follows:

(1) Patients affected with chronic nephritis and under observation for a long period eliminated less solids and less urea than normal individuals.

(2) Those who died from uremia excreted still less solids and urea, than those who did not succumb to the disease.

(3) The deficiency in urinary excretion is greater in chronic interstitial, than in diffuse parenchymatous, nephritis.

These facts in my opinion are a strong support to the theory, which ascribes the severest symptoms of nephritis to a retention of substances normally excreted by the kidneys, and do not support the new doctrine of nephrolysins and internal renal secretion. These teachings have also been opposed by the researches of many investigators, such as Bedano and Tarchetti, Ciotti, Albarran and Bernard, Lemoine and Linossier, Ceconi and Micheli, as well as by the thor-

ough work of Stern, of Geneva. The important discussion at the Clinical Society in London in which Lauder Brunton, Horsley, Bradford and others disputed most emphatically the theory of the existence of an internal renal secretion is also worthy of mention. Of considerable importance further is the communication of Achard and Poisseau to the "Société de Biologie" at Paris, stating that they had observed changes in the convoluted tubules upon the injections of extracts of fresh organs and hypotonic solutions.

I should not have touched upon this subject, were it not that some authors credit the benefits of decapsulation and nephrotomy in nephritic processes to the elimination of the nephrolysins, and were it not that physicians believing in these new doctrines are prescribing large quantities of raw kidney for their poor patients and are formulating strange theories based upon an hypothesis not well established in science.

In my opinion, however, as well as in that of most of the men who have busied themselves with the subject, the improvement and recovery from the nephritic process after decortication is due to the modification of the blood circulation, to the better irrigation of the diseased organ with blood.

That the irrigation of the renal glomeruli and tubules has great influence on the function of the kidneys also becomes evident from the table of Dr. Tino; it illustrates very clearly the well-known fact, that less solids and less urea are eliminated and severe uremic symptoms are observed more frequently in interstitial nephritis than in chronic parenchymatous and diffuse nephritis; this is due to more marked blood-vessel changes in the former.

My own experiments, as well as those of other authors, show, moreover, that incision of the renal capsule soon diminishes both the increased tension of the organ and the engorgement of its blood vessels, frequently causing a copious discharge of blood and perhaps also of toxic materials and epithelial detritus, which has accumulated in the kidney. Thus, the first effect of decortication is an increased flow of urine, a diminution of stasis, so deleterious to the life of the tissues, and the restoration of their functions.

Claude and Balthazard have shown that decapsulation of the kidney in the dog induces a more complete urinary depuration with less work on the part of the heart, and from this they conclude

that the same operation in man diminishes vascular tension. Baccelli, some time ago, expressed the view, that no matter what the cause inducing the nephritis, and more particularly a glomerulonephritis, the greatest damage is due to venous congestion; he therefore recommended venesection at the foot as the best means of relieving this congestion, and practiced it with good results.

Decortication also exerts a favorable influence on the course of the nephritis by the formation of new blood vessels; thus the circulation impaired by the inflammatory process is made freer, the molecular changes in the diseased tissues become more active, and the healing process is enhanced. Edebohls in his patients operated upon observed an almost immediate increase in the flow of urine, an increased elimination of urea, and an improvement in their general condition, such as disappearance of the edema, headache, gastric disturbances, and a perfect restoration of strength and color of the skin. The two postmortem examinations by Jawelt and Cutter on patients dying a considerable time after decapsulation of the kidneys fully confirmed the experimental results obtained by myself and others, such as Balthazard and Claude, Johnson, Albarrard and Bernard, Gayet and Bosson, Anzilotti and Ferrarini, etc. They found a newly formed renal capsule and vascularized adhesions, which serve to establish new blood paths between the kidneys and the adjacent tissues. Edebohls himself confirmed these conditions recently at autopsies on patients dying respectively four and five months after the operation.

The fear that the new capsule might produce phenomena of compression and later renal sclerosis was opposed by Edebohls, and has been found groundless by the experiments of Anzilotti—who observed no such tendency five months after the operation. It was also proved groundless by certain clinical observations which showed good health and normal urine to exist in patients even 10 years after the date of the operation.

Thus, Harrison's theory, according to which capsulectomy favorably influences the nephritic process by diminishing the internal tension of the inflamed kidney, and Edebohls's theory, according to which the beneficial results are due to the new blood paths between the kidneys and the neighboring organs, complement each other, and are in my opinion supported by clinical and experimental evidence.

From these facts I consider myself justified in outlining the following indications for the performance of decortication of the kidneys: It should be performed: (*a*) in cases of acute nephritis; (*b*) in cases in which anuria and subsequent uremia supervene in the course of chronic nephritis; (*c*) in cases in which the patients are still young and robust and there is imminent danger of losing life; and (*d*) in cases in which we have tried all the medical measures at our command without obtaining success. The operation will at least hold out a hope of saving our patients from an otherwise unavoidable death. In maintaining this view I am in accord with the opinions expressed by Morgour, Claude, Edebohls, Pousson, Guiteras, and other clinicians, who state that anuria, nephralgia, hematuria, and in general all those physical and functional symptoms, which by their persistence render death probable and are obstinate to medical treatment, can be successfully treated by surgical intervention.

DISORDERS OF THE UMBILICUS, WITH SPECIAL REFERENCE TO THE NEW-BORN AND THE INFANT: I. UMBILICAL HERNIAS

BY A. ERNEST GALLANT, M.D.

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In a paper read before the American Medical Association on "The Antenatal and Intranatal Factors in Neonatal Pathology," J. W. Ballantyne,[1] of Edinburgh, concludes that "it is clearly evident that if the characters of the new-born infant are to be understood it is essential that account be taken not only of the facts that the infant's organism has just passed through a period of traumatism and is passing through one of readjustment to meet the new requirements, but that also during the nine months of intrauterine life which preceded birth, it may have been the sphere of morbid processes which have left their imprint on it. It may come into the extrauterine environment diseased or malformed or predisposed to some pathological development. Like pregnancy, neonatal life is an epoch which has a physiology in many respects peculiar to itself, and which borders very closely on the pathological, tending very easily to pass over into it. In a certain sense the ordinary vomiting of pregnancy is to the uncontrollable form as the ordinary 'physiological' (?) jaundice of the new-born is to pernicious icterus neonatorum. Further, just as every woman brings with her into her pregnancy the results of her past pathological history, so the new-born infant brings with him, out of his antenatal into his neonatal existence, the effects of any morbid process which may have attacked him in utero. In this way the pathology of pregnancy and the

[1] Jour. Amer. Med. Assn., 1899, xxxiii, 1249.

maladies of the new-born infant are invested with peculiarities. *The peculiarities, therefore, of neonatal diseases are not inexplicable, but are the direct outcome of antenatal and intranatal factors on the organism of this period of life.*"

At the Good Samaritan Dispensary, from 1887 to 1896, the total number of children, ten years and under, treated was 254,036. Of this number 18,042 were referred to the late Professor Van Arsdale for surgical care. From his records I collect the following anomalies of the umbilicus:

	Cases
Acquired hernia, under one year (youngest 3 weeks)	145
One to 5 years	66
Six to 10 years	6
Total	217
Acquired hernia and hydrocele, at two months	1
Inguinal hernia, at three months	1
Phimosis, at six months	3
Fungus, at three months	1
Syphilis, at three months	1
Umbilical fungus (with or without infection of the arteries and veins)	135
" " and hydrocele	1
" abscess	1
" fistula	1
" hemorrhage	2
Ectropion vesicæ	1

With this experience in mind, it is my purpose briefly to pass in review the origin and development of the structures involved in the formation of the umbilicus; the manner in which these may deviate from the normal; and the diseases incident thereto, with especial reference to those which cripple or imperil the life of the child at birth and during infancy.

DEVELOPMENT OF THE UMBILICUS

At the end of the second week of fetal life the embryo curves upon itself both longitudinally and laterally, so as to resemble "a turned-over canoe, the well of the canoe,—that is, the body-cavity of the embryo, opening into the cavity of the yolk-sac, a portion of which now lies within the body-cavity, and a portion outside it, the two portions communicating by a duct, the omphalo-mesenteric

duct. From the edges of the embryo there emerge arteries and veins, which pass through the duct to the outer portion of the yolk-sac, which is now termed the umbilical vesicle and provides nutrition to the embryo until such times as the allantois is formed."

The intra-abdominal portion of the yolk-sac develops to form the entire intestinal canal. From its caudal extremity shoots forth a diverticulum which projects outward between the abdominal plates and forms a sac known as the allantois or chorion, from which are derived the blood-vessels and tissues making up the placenta. From the intra-abdominal portion of this intestinal branch are developed the urethra, bladder, and a remnant known as the urachus.

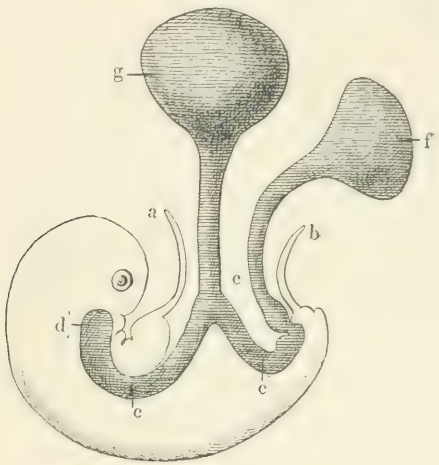

FIG. 1.—Formation of the alimentary canal. *a*, *b*, commencement of the amnion; *c*, intestine; *d*, pharynx; *e*, urinary bladder; *f*, allantois or chorion; *g*, umbilical vesicle. (Redrawn after Dalton.)

The structures involved in the formation of the umbilicus are: (1) The abdominal plates, which unite along the median line to enclose the viscera within the abdominal cavity. (2) The omphalomesenteric duct and the omphalomesenteric arteries and vein, derived from the umbilical vesicle. Normally these structures undergo retrograde changes, and leave no remnant. (3) The allantois and its vessels, from which are derived the umbilical cord, extra-abdominally, and the urethra, bladder, and urachus, intra-abdominally. (Figs. 1 and 2.)

The Abdominal Plates.—When the abdominal plates fail to unite during the early weeks of fetal life, the colon, intestines,

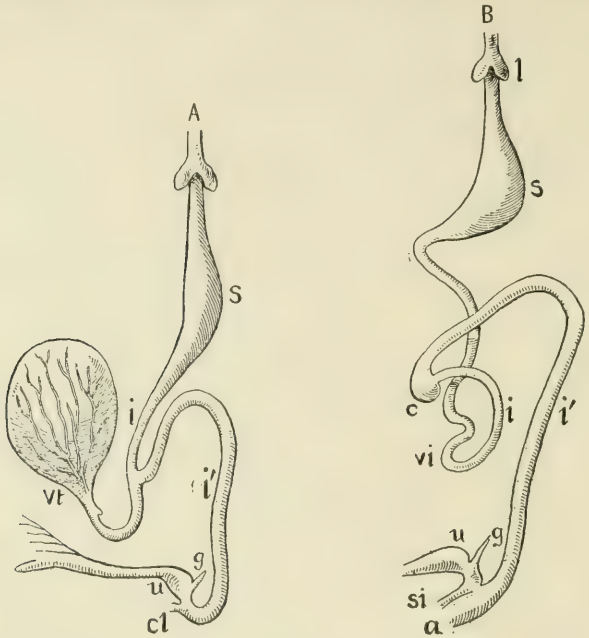

Fig. 2.—Outlines of the form and position of the alimentary canal in successive stages of its development. *A*, alimentary canal, in an embryo of four weeks; *B*, at six weeks; *i*, small intestine; *i'*, the large intestine; *c*, the cecum; *r*, the rectum; *a* (in *B*), the anus distinct from *st* the sinus urogenitalis; *v*, the yolk-sac; *vt*, the vitello-intestinal duct; *u*, the urinary bladder and urachus leading to the allantois; *g*, genital ducts. (Redrawn after Allen Thomson.)

Fig. 3.—Fetal pig, showing a protruding loop of intestine, forming an umbilical hernia. From the convexity of the loop a thin filament is seen passing to the umbilical vesicle, which, in the pig, has a flattened, leaf-like form. (Redrawn after Dalton.)

appendix, cecum, right kidney, and liver, one or all, covered only by the thin, transparent amnion, may protrude at the umbilicus,

giving rise to a hernia into the cord, a *true congenital hernia.* (Figs. 3, 4, and 5.)

After cicatrization of the umbilicus, the intestine or omentum or both, covered by an opaque layer of skin and peritoneum, may protrude through the umbilical ring—*acquired umbilical hernia.* (Fig. 6.)

FIG. 4.—Pedunculated fetal hernia, containing intestines only. (Redrawn after Chadwick.)

The cord may be attached to the ring after an abnormal fashion —*amniotic umbilicus.*

THE OMPHALOMESENTERIC DUCT.—Incomplete retrograde changes in the abdominal portion of the vitelline duct may result in:

(*a*) A cul-de-sac, opening into the ileum—*Meckel's diverticulum.*

(*b*) A cul-de-sac, opening at the umbilicus—*a mucous sinus.*

(*c*) An open tract, from the ileum to the umbilicus—*fecal fistula;* frequently associated with eversion and prolapse of the abnormally exposed mucous membrane—*adenoma funiculi.*

(*d*) A band stretching from the small intestine to the umbilicus or elsewhere—of importance as the cause of intestinal obstruction.

The Urachus.—A remnant of the allantois, stretching from

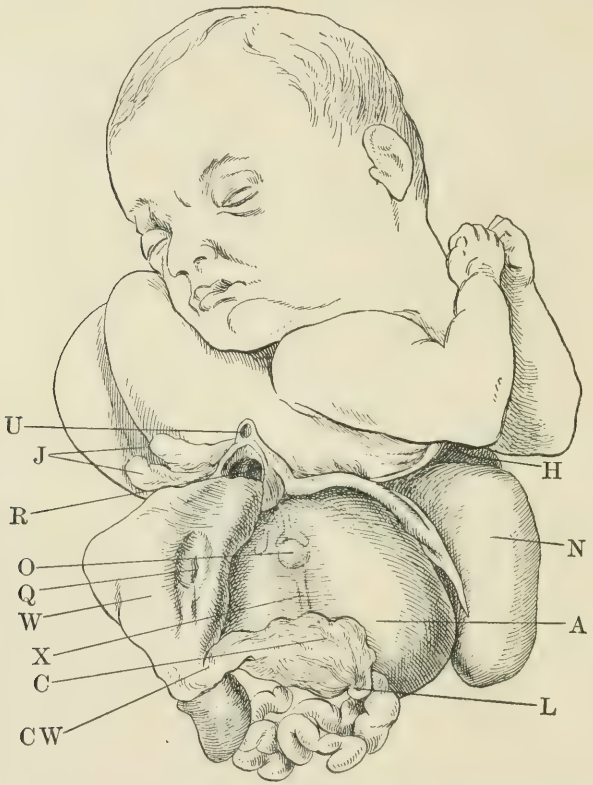

Fig. 5.—Fetus with abdominal cleft through which protrude the heart, left lung, and all the abdominal and pelvic organs, besides a large cyst. The head rests on the buttocks owing to extreme flexion of the vertebral column. H, heart; N, liver; A, cyst; I, ileocecal valve; J, labia majora; U, umbilical vein; R, undeveloped uterus (?); O, right ovary; Q, pouch from the bladder; W, exstrophied bladder; X, vena cava inferior; C, colon; CW, junction of colon and bladder. (Redrawn after Chadwick.)

the fundus of the bladder to the umbilicus, extraperitoneally, may remain patent in any portion of its length and form.

(*a*) A cul-de-sac communicating with the interior of the bladder—the *vesicle diverticulum* of Schullenback.

(*b*) A cul-de-sac opening at the umbilicus—the *sebaceous sinus* of Van Derveer.

(*c*) The canal remains patent, or becomes so later—*vesico-umbilical urinary fistula.*

(*d*) The urachus, closed at both ends, becomes distended with fluid, forming one or more sacs—*cysts of the urachus, allantoic cysts,* or *tubulo-cysts* of Bland Sutton.

The protruding intestine or stomach may be included when tying the cord, and result in a fecal or gastric fistula.

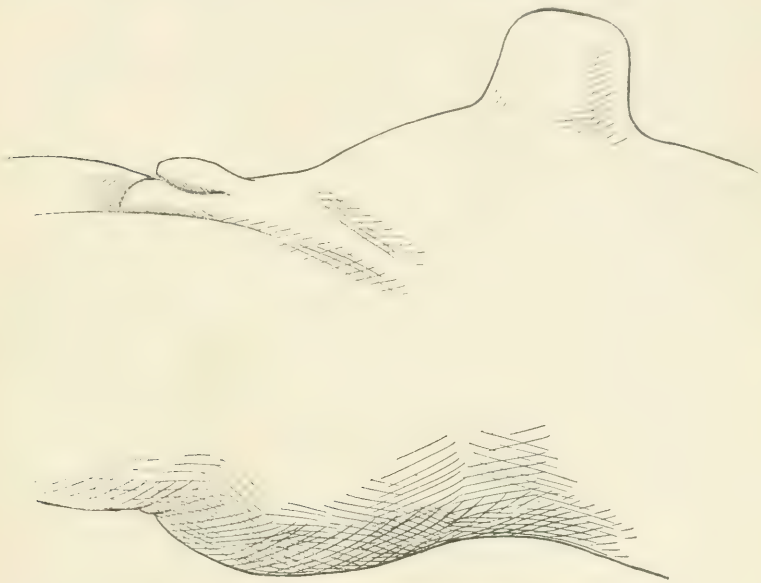

FIG. 6.—Acquired umbilical hernia.

Infection of the umbilical stump may involve the vein, the arteries, or both—and this may be manifested by biliary fistula, hemorrhagic fistula, jaundice, or the escape of pus from the umbilicus.

Neoplasms develop from the various tissues which go to make up the umbilicus.

While the above classification accounts for the anatomic features involved in the origin of anomalies arising at the umbilicus, for practical purposes I deem it better to group them under the following headings: (1) Hernias; (2) fistulas, sinuses, and cysts; (3) infections; and (4) neoplasms.

ANOMALIES ARISING AT THE UMBILICUS

A. Manifest before the cord separates.

1. Hernia into the cord—congenital hernia.
2. Amniotic umbilicus.
3. Hematoma.
4. Angioma.

B. Manifest after the cord separates.

	Fistula.	*Sinuses.*	*Cysts.*
5. From vitteline duct.		Mucous (polypi).	
6. From allantois (urachus).	Fecal (adenoma). Urinary.	Sebaceous. Dermoid.	Tubulo-cysts.
7*a*. Inclusion intestine.	Fecal, with adenoma opening above and below.		
7*b*. Inclusion stomach when tying cord.	Gastric, opening into stomach.		
8. Infection of umbilical veins and arteries.	Biliary, with or without jaundice. Hemorrhage, with bleeding, or pus or both.		

9. Through patent umbilical ring—acquired hernia: omentum or intestine or both.
10. Neoplasma. (*a*) Granuloma; (*b*) lipoma; (*c*) fibroma; (*d*) sarcoma; (*e*) myxo-sarcoma; (*f*) epithelioma.

UMBILICAL HERNIA

In the new-born and in infants, protrusion of one or more of the abdominal viscera is recognized as of two varieties: (*a*) congenital, occurring before birth; and (*b*) acquired, that which takes place after cicatrization of the umbilicus.

CONGENITAL UMBILICAL HERNIA: HERNIA INTO THE UMBILICAL CORD

"After the small intestine is formed, it increases rapidly in length; it grows, for a time, faster than the walls of the abdomen; and as it can no longer be contained in the abdominal cavity, it protrudes under the form of a hernia, from the umbilical opening. (Fig. 3.) In the human embryo this protrusion continues during the latter part of the second month. Subsequently the walls of the abdomen grow more rapidly than the intestines; and, gradually enveloping the hernial protrusion, at last enclose it in the cavity of the abdomen." [2]

[2] Dalton. Human Physiology, Seventh Ed., p. 674.

"The omphalomesenteric duct is usually patulous until about the end of the fourth week of fetal life. It then rapidly shrivels to a thin cord and disappears by the end of the sixth week, when the intestine retreats into the abdominal cavity, and the umbilical ring closes. The remains of the duct in the umbilical cord may be seen as a slender pedicle as late as the end of the third month, and vestiges of it—and even of the vesicle—have recently been discovered in many placentæ and cords, after delivery at full term." (Chadwick, quoting Schultze.) [3]

Three factors are held to be responsible for the production of congenital hernia: (1) Deficient or delayed development of the abdominal walls, which fail to unite, or but partially coalesce, and result in an opening through which the intestines and other abdominal viscera protrude—covered only by the transparent chorion and Wharton's jelly. (2) The umbilical vesicle and its duct, with their vessels, fail to atrophy in the process of embryonic growth and persist, to a later period than usual, as a cord anchoring that portion of the intestinal tube with which they are in connection outside the umbilical ring. This form may be distinguished by the fact that the hernial sac lies at a distance from the umbilicus, being connected with it by a neck or pedicle (Chadwick). (Fig. 4.) Foerester refers to the frequent coincidence with umbilical hernia of anterior curvature of the vertebral column, so that the body appears to be bent at its middle. One of Chadwick's cases is a remarkable instance of the extent to which this curvature may be carried, if the traction upon the middle of the column be greatly increased by an adventitious growth. (Fig. 5.) Spina bifida was present in five of the six specimens studied by Chadwick. (3) Intrauterine peritonitis may result in adhesions of the viscera one to the other, and to the amniotic covering, and prevent their recession within the abdominal cavity. (Simpson.[4]) "The umbilical herniæ which develop later in fetal life and are usually smaller may also arise from the fact that by compression and malposition of the fetus within the uterus a peritoneal sac is formed at the umbilicus, into which intestine may enter" (Tillmanns).

The sac in congenital hernia may contain any or nearly all of

[3] Trans. Amer. Gyn. Soc., 1876, i. 367.

[4] Edinb. Med. and Soc. Jour., Oct., 1838, p. 390.

the abdominal viscera, including the stomach, large and small intestines, cecum and appendix, liver, and right kidney.

The *size of the hernial tumor* depends upon the number of viscera within the cord, and has been variously described as large as a walnut, hen's egg, chicken's egg, the fist, a quart measure, etc.

The *transparency of the sac* permits of a ready recognition of the viscera therein, and a definite diagnosis.

Rupture of the sac during labor occurred in the hands of Piering [5] with protrusion of the intestines. The condition was cured by operation during the first hour of life.

The Treatment of Congenital Hernia.—That mechanical measures may result in curing some cases even of large size is shown by Lobstein's case, in 1768,[6] which healed in four weeks under a simple protective dressing (reduction of the liver); and Storch's [7] case in 1751 (repression of the "apple-sized" hernia), with compression by means of strips of plaster in form of a cross; child being alive 14 years later. Ligature of sac and hare-lip pins was successfully practised in 1804, by James Hamilton,[8] as a means of closing the aperture after reducing the prolapsed intestine. Suture of the aperture, according to A. O. Lindfors' first table [9] was first carried out by Berard in 1836; after dissecting off the sac the edges of the skin were sutured together, and the child cured in 3 weeks.

To the unknown House-Surgeon of the Ferrara Hospital belongs the credit of being the first to freshen the wound before suturing, an honor paid him by Cruveilheir [10] who in 1849 saw and related the circumstances under which he observed the case. Reuterman [11] in 1861, 14 hours after birth, reduced a hernia as large as a child's head, made up of liver and intestines, closed the opening with a ligature, and cured the child in 27 days.

Radical Operation.—Doran [12] in reviewing 13 cases collated

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[5] Amer. Jour. Obstet., 1898, 37, 794.

[6] Bucholz: Dissertatio de Hepatomphalocete.

[7] Therapeut. und Prakalkund v. Kinder, Eisenbach, 1751, 4, B, 35S.

[8] Anatomy and Surgical Treatment of Crural and Umbilical Hernia, 1807, p. 57.

[9] Hygiea, xliv, 1–4.

[10] Path. Gen., 1849, i, 628.

[11] Reuterman. Hygiea, 1861, xxiii.

[12] Lond. Med. Recorder, Aug. 20, 1889, 131 and 312.

by Lindfors claims for Treves[13] the credit of having been the first to do a radical operation for congenital hernia. Treves incised the whole length of the sac in the median line, enlarged the abdominal opening, relieved intestinal distention by the introduction of a hypodermatic needle, released 8 inches of small intestine, inserted hare-lip pins, through and including the peritoneum, and sutured the skin with silk.

To Lindfors,[14] without doubt, we must render honor as the first to insist on herniotomy under antiseptic precautions: the closure of the abdominal cavity entirely by suture in tiers and by his analysis of the 77 cases[15] tabulated, the enforcement of the excellent results secured by early radical operation.

Adherent irreducible, congenital hernia has been met with at or soon after birth. D'Arcy Power[16] has reported two fatal cases which he attributed to this condition. From his experience he believes that: (*a*) These cases are not frequently met with; (*b*) that owing to the decomposing covering, adhesion of the sac soon takes place, even if this condition was not present before birth; (*c*) that waiting will result in strangulation, and is unwise; (*d*) that as soon as the diagnosis is made, taxis should be immediately followed by abdominal section; and (*e*) that when vomiting as a sign of peritonitis is present, the prognosis is bad.

Doran[17] concludes: (*a*) That the new-born infant bears abdominal section very well; (*b*) that cutting seems less dangerous than cold (?); (*c*) that operation may be done with or without anesthesia, or an anesthetic may be given as early as one hour after birth; (*d*) that when discovered in the new-born it is clear that the right treatment, except when the hernia is very large and the deficiency of the parietes great, is to perform herniotomy as soon as possible.

H. E. Safford,[18] from a careful analysis of the cases tabulated by Lindfors, Berger, Hallet, and elsewhere, found that as the result of radical operation: Laparotomy, freshening the wound edges and

[13] Lond. Lancet, 1881, i, 323.
[14] Loc. cit.
[15] Volkmann's Sammlung Klin. Vortrage, N. F., No. 63, 1893.
[16] Lond. Lancet, 1894, ii, 1216.
[17] Loc. cit.
[18] Phila. Med. Jour., 1901, vii, 393.

suturing, 65.6 per cent. healed; while under expectant treatment but 47 per cent. healed. Hallet (quoted by Safford) concludes that: "Intervention should be as early as possible after birth; waiting endangers the membrane from gangrene and peritonitis; and radical operation is always the most rational if we have the means and assistance at hand." His summary shows that—

In 58 cases operated upon within 36 hours after birth.....	15 deaths	(26%)
In 7 cases, within 48 hours.....	3 deaths	(43%)
In 2 cases, on third day.....	1 death	(50%)
In 4 cases, after third day.....	4 deaths	(100%)

Referring to Safford's tables we note 32 patients operated upon during the first 24 hours and find that but 6, or 18.75 per cent., died, a very strong argument for immediate interference. Of those operated upon the first five hours, all (8 patients) lived.

The *operative technic* is that of umbilical hernia of later years: (*a*) Chloroform anesthesia; (*b*) reduction of the contents, or if irreducible, (*c*) incise sac, enlarge opening along the median line, if necessary, (*d*) separate adhesions and complete reduction, (*e*) cut away sac and ligate vessels, and suture layer by layer. Olshausen [19] dissects around the margin down to but without opening the peritoneum, removes Wharton's jelly, sutures the peritoneum into folds, and covers the whole mass by bringing the abdominal walls together with sutures. The 5 patients operated on by this method lived but protrusion at the navel occurred later.

ACQUIRED UMBILICAL HERNIA IN INFANTS

After the umbilicus has healed the ring may fail to contract and close, the cicatrix give way, and the omentum or intestine or both protrude through the ring. In this class of cases (acquired hernia) the opaque sac is composed of peritoneum covered only by skin, through which the contents of the sac cannot be seen, but are readily recognized by palpation.

Owen maintains "that on seeing an umbilical hernia the first thing to be done is to find out why the protrusion has occurred. It may be that it was acquired because the infant had been perpetually straining with vomiting, in which case, if a flat pad is strapped over the aperture and the food and feeding are properly

[19] Archiv f. Gynæk., 1887, xxix, 443.

regulated, a cure may be gradually effected. Sometimes, again, the hernia is affected by straining set up by a tight foreskin, by diarrhea, constipation, a rectal polypus, or by whooping-cough. In such cases a radical operation is out of the question. Then, again, in a rickety child, when the bowels are distended by the accumulation of gases and the linea alba is frayed out and the interval between the recti abdominalis is widened, the umbilical scar must needs be weakened and the occurrence of an exomphalos is invited. Under these circumstances the administration of repeated doses of rhubarb and soda, together with careful dieting and supervision, may effect all that is needed. But when the causes of the rupture are obscure, when no clear indications for treatment are discoverable, and when the abdominal aperture is large and the flat pad and strapping have failed, a radical operation is not only justifiable, but necessary.

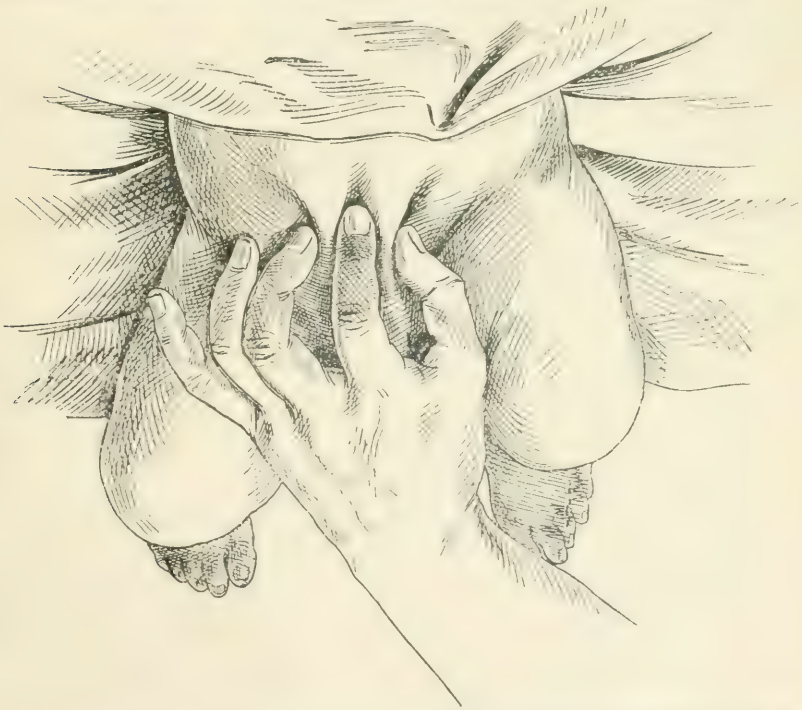

FIG. 7.—The method of invaginating the sac of an umbilical hernia and approximating the recti muscles so as to cover in the umbilicus.

THE TREATMENT OF ACQUIRED UMBILICAL HERNIA.—In the service of the late Professor Van Arsdale at the Good Samaritan

Dispensary, of the 217 cases above mentioned, 3 were referred to the hospital for radical treatment. To the remaining 214 cases compression was applied in the following manner: (*a*) Reduction of the contents of the sac by careful manipulation; (*b*) inversion of the sac and approximation of the recti and abdominal wall so as to cover in the umbilicus (Fig. 7); (*c*) the application of a pad, silver dollar size, made of thick cardboard, covered with adhesive plaster, *sticky side out,* directly over the site of the hernial opening; and (*d*) this pad secured *in situ* by a strip of "Z O" plaster 3 x 6 inches, placed across the abdomen (Fig. 8). This dressing was

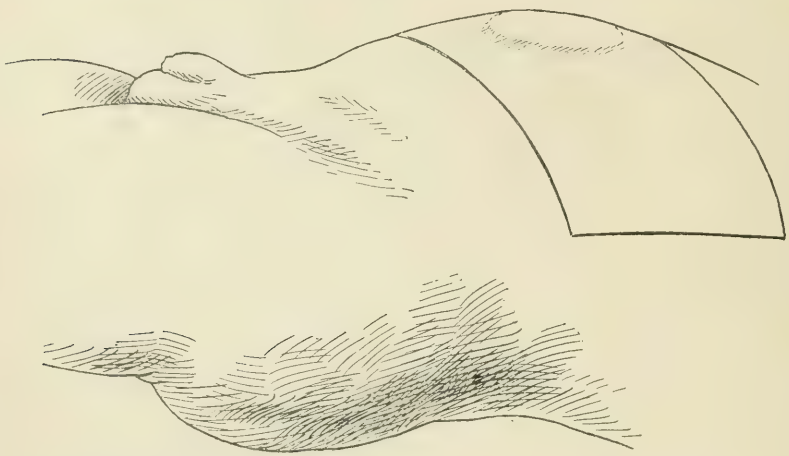

FIG. 8.—Method of applying a card-board pad (silver dollar size) to retain an umbilical hernia.

renewed every seven to ten days, and though worn for months rarely caused irritation of the skin. This inversion of the sac and approximation of the recti, preventing the gut from at any time protruding through under the pad, was considered the most important factor in securing a rapid contraction of the opening and an early cure.

"The use of a ball or the conical pad usually employed is a serious mistake, as it presses into and dilates the ring, aggravating the very object for the cure of which it is employed." [20]

Prognosis.—Coley [21] tells us that "this form of hernia (ac-

[20] Dallas, Internat. Jour. Surgery, Feb. and April, 1892.
[21] Sajous's Annual, 1899, iii, 432, 440.

quired) though frequently met with, never leads to strangulation, and quickly subsides by contraction of the opening if, after reduction, appropriate measures are resorted to;" and that "umbilical hernia in children should, with rare exception, never be operated upon, for the reason that they are almost invariably cured either spontaneously or by means of mechanical support." . . . Cases over four years of age, in which mechanical treatment has been faithfully tried for a number of years without benefit, should be closed by operation, by the Bassini method.

Obstetrics and Gynecology

FETAL HYDROCEPHALUS AS A CAUSE OF DELAY IN LABOR

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Increase in size of the fetus is equivalent, as regards its effects, to diminution in the size of the maternal pelvis. Further, a labor which is delayed by reason of a pathologic enlargement of the infant is apt to have more serious effects than one in which the cause of the delay is found in the maternal canals, hard or soft; and the reason is that in the latter case the cause is obvious, or at least easily and early recognized, while in the former it is often quite unsuspected until the confinement has been many hours in progress. The fetal enlargement may be general, as in the cases of postmature labor, and in these circumstances delivery is apt to be long delayed and accompanied by grave risks, as I have elsewhere shown and illustrated.[1] It may, on the other hand, be localized to the trunk or head, as in fetal ascites or fetal hydrocephalus, and, then, if the cause of the dystocia be recognized in time, the labor can usually be safely and quickly terminated by means of appropriate treatment. I have within the past eighteen months had three cases of labor complicated by fetal hydrocephalus under my care; in two of them the infant was brought down or presented by the breech, and in both of these instances delivery was effected by tapping the fetal spinal canal, while in the third case the labor was complicated by maternal eclampsia and the hydrocephalic fetus came by the head. It is more particularly to the first and second cases and to the mode of

[1] Jour. Obstet. Gynec. Brit. Emp., 1902, ii, p. 521.

operative interference adopted in them that I wish to draw the attention of the reader.

Case I.—On August 15, 1901, a woman was sent into the Royal Maternity Hospital with her infant born as far as the shoulders, the fetal head being still above the brim of the pelvis. The patient had had three previous labors: the first and third had been normal and the children were now alive and well—a girl and a boy; the second had ended in the birth of a dead female fetus about whose condition no further details were to be obtained. The present pregnancy (the fourth) had been rendered troublesome by pain in the left side and by unusual distention of the abdomen. Labor pains began on the evening of August 14 and the patient's medical attendant was summoned the next morning; he diagnosed a transverse presentation and in the afternoon (the os being now fully dilated) he ruptured the membranes and performed podalic version. The feet were easily brought down, but the arms and trunk only after considerable traction had been made. It was then discovered that no force could extract the head. Another medical man was summoned, but still the head remained fixed above the pelvic brim. At first twins had been suspected, but now the physicians began to think of a fetal malformation; there was no contraction of the mother's pelvis. It was, therefore, decided to send the patient into hospital.

I saw her soon after her arrival at the hospital. She was in a somewhat collapsed state, her pulse being 140 and weak and her lips and face pale. On abdominal palpation I felt the uterus fairly well contracted and of the size it usually is during the first stage of labor; but from the vulva there protruded the trunk and limbs of a male infant, the back being to the right side. More careful palpation revealed a regular elastic swelling in the uterus which yielded a slightly crackling sensation in the lower part. By vaginal examination I felt a well-ossified occiput with a membranous part beyond it. The diagnosis therefore narrowed itself down to hydrocephalus or to a still rarer condition of encephalocele or meningocele or congenital cranial tumor. The umbilical cord, it may be added, was pulseless.

I resolved to treat the case as if it were one of fetal hydrocephalus; so by gentle manipulation I brought the back of the child

round to the front of the mother's pelvis, and with a knife made a short transverse incision over the interspace between the sixth and seventh dorsal vertebræ cutting into the spinal canal. I then passed a long silver catheter up the canal and into the cranial cavity until I felt its tip moving freely inside. About 36 ounces of clear fluid then came away through the tube. Immediately thereafter I was able by means of slight traction (one of the house surgeons aiding me with suprapubic pressure) to draw the fetus out of the passages. It was a male and had no other malformations than the hydrocephalus. With the fluid which had been drawn off it weighed 12 pounds; the cranial bones were well ossified but were separated from one another by wide membranous spaces. The placenta weighed 36 ounces. During the first 24 hours after labor the mother's temperature was 100° F., and her pulse varied from 150 (immediately after labor) to 104. There was a quick pulse and a temperature varying from 100° to normal for a few days; but the patient made a complete recovery, and left the hospital well on the fifteenth day.

Remarks.—In this patient's case the infant presented by the shoulder, and it was only after version had been done that it was discovered that there was a cause of delay existing in the child itself. A great deal of interference had taken place before the woman was sent into the hospital. In addition to the version, there had been the bringing down of the arms, the frequent vaginal manipulations, the prolonged traction, and two abortive attempts at the application of forceps. It seemed to me, therefore, that any method of delivering the woman which avoided further intravaginal and intrauterine handling was to be preferred. Of course the hydrocephalic head could have been tapped, as it lay above the brim of the pelvis; but spinal paracentesis was far better, for it quickly removed the obstruction to delivery (an important matter when uterine rupture is feared) and did so by an operation which was entirely extravaginal. To the procedure itself I shall draw attention after I have described the second case.

Case II.—On November 28, 1905, I was sent for to help a medical friend (Dr. Millard, of Leith) with a difficult case of labor. The patient was a woman, 46 years of age, and the labor was her fourth. Ten years ago she had her first child; in that case the

FIG. 1.—Rear view of the fetus, showing the hydrocephalus and the spina bifida (case II).

FIG. 2.—Front view of the hydrocephalic fetus (Case II).

labor supervened six weeks before full term, and the infant, it was said, had been dead for some weeks before its birth. The child was a female. The second pregnancy was accompanied by excessive vomiting. Fetal movements were first felt at the beginning of the seventh month and labor came on a week later; there was marked hydramnios. The fetus—a female—was extracted with forceps, and turned out to be anencephalic; and the placenta was entirely adherent and had to be removed manually. The fetus showed, in addition to the anencephaly, an open state of the spinal canal and (on dissection) a well marked diaphragmatic hernia. It was described by me in a now defunct journal (The Physician and Surgeon, vol. i, p. 891, 1900). The third pregnancy ended in the birth of a healthy living male child; the labor was somewhat delayed, partly on account of some contraction of the mother's pelvis which existed and partly because the position of the head was occipito-posterior. This birth was in 1900 and the boy is now alive, well, and shows no deformities or signs of hydrocephalus. The present pregnancy, the fourth, had been accompanied by bad health, the patient having been unfit for her ordinary household duties. Labor came on at the full term, but the pains were ineffective. The breech was felt at the pelvic brim, but it did not at first engage. When I arrived I found that some progress had taken place, the breech being now at the vulva. I managed, with some difficulty, to bring down the legs and trunk, and with still greater difficulty to release the arms which had slipped up alongside the head, above the contracted pelvic brim. I then noticed that the infant was a female and that there was a spina bifida in the lumbar region (Fig. 1). Abdominal palpation revealed a greatly distended uterus in which no hard fetal head was to be felt, but rather a general, even, rounded swelling in which fluctuation could be indistinctly made out. With the former history of the patient in my mind, I at once thought of some fetal malformation; and with the large size of the uterine swelling and the presence of the spina bifida before me, I could hardly fail to diagnose fetal hydrocephalus. I adopted the same method of treatment which I had used in the Maternity Hospital a year ago; but in this instance I resolved to utilize the opening of the spina bifida for the passing of the catheter into the cranial cavity.

Accordingly I passed a male silver catheter (No. 6) up the

spinal canal and moved the point of it about inside the cranium. In a few seconds a clear fluid began to flow from it, and soon Dr. Millard, whose hands were on the abdomen, said he could distinctly feel the separate bones of the collapsing fetal head. Traction on the fetal trunk now served to complete the delivery, there being only slight resistance at the contracted brim. The placenta came away a few minutes later but the membranes were adherent and had to be removed manually. The patient made a good recovery.

The infant, a female, weighed (with the head redistended with water) more than 10 pounds (Fig. 2). The circumference of the head in the occipito-frontal plane was 23 inches. The placenta was large and thin. It is noteworthy that in this woman's previous labor (which ended in the birth of an anencephalic fetus) the placenta had the same character. Dissection of the fetus revealed no diaphragmatic hernia or defect.

Remarks.—This case, besides demonstrating in an excellent manner the advantages of spinal tapping, afforded a striking instance of recurrent monstriparity. The mother gave birth to four children, of whom three were females and one a male. Of the three females, one was born dead (whether deformed or not could not be ascertained), one was anencephalic, and one was hydrocephalic; the male alone was healthy, well formed, and survived his birth. This phenomenon is by no means infrequent in the annals of teratology, and I have put on record several examples of it, of which perhaps the most striking was that reported in the American Journal of Obstetrics in 1900 (vol. xli, p. 577). In that case a woman gave birth to three malformed fetuses. One was a hydrocephalic female fetus with absence of the right thumb and radius; another was an anencephalic female fetus with a malformed thumb and other anomalies; and the third was a hydrocephalic female fetus with absence of the right thumb, both radii, and a malformed state of the left thumb. The mother herself, as well as a sister and her mother, had defective muscles of the thumb. The male infants in this case, as in the one now recorded, were healthy and well formed. I have elsewhere dwelt at some length upon the bearing of recurrent monstriparity upon the problems of teratology.[2]

[2] Jour. of Obstet. Gynæc. Brit. Emp., ix, III, 1906.

SPINAL TAPPING IN HYDROCEPHALUS

The two cases which have been recorded furnish excellent examples of the method of delivering the infant in cases of hydrocephalus which seems first to have been proposed by J. Van Hoevel in 1848 [3] and first to have been carried out by Tarnier in 1865.[4] It is claimed, however, by Hubert [5] that Lecoux was the first to establish it as a useful obstetric operation. At any rate there are indications that Tarnier's assistants and pupils were in the habit of employing the operation as opportunity arose. N. Charles, of Liège,[6] used the method successfully in 1881. In the hands of Oui [7] the maneuver failed, probably because a fracture of the cervical spine with separation of the bones prevented the passage of the catheter. Ira G. Stone [8] and B. Pozzoli [9] reported cases toward the close of the nineteenth century. My case reported in 1904 [10] seems to have been the first recorded in Great Britain, although Dr. J. Dougall [11] recommended its employment as early as 1881 in the cases where there was a spina bifida as well. Dougall did not, however, advise anything more than the opening of the spina bifida, as the following quotation from his paper proves:

"In this case" (one of fetal hydrocephalus presenting by the breech and complicated by spina bifida) "had the labor not finished when it did, I would have used the perforator. It occurred to me after, that I had neglected an opportunity of making one of the complications obviate the other. This could have been done after the child was dead, by opening the tumor at the bifid spine, and so tapping the cranium through the cerebrospinal canal. This operation might probably be performed in lieu of craniotomy in fetal hydrocephalus, even where the spine is not bifid. An incision could be made into the cerebrospinal canal about the dorsal region, after turning, on in a breech presentation, and the cranium emptied of its superfluous fluid thereby."

[3] Presse Méd. Belge, i, 279, 343, 1848–9.
[4] Traité de l'Art des Accouchements, iv, 32, 1901.
[5] Cours d'Accouchements, ii, 254, 268, 1878.
[6] Jour. d'Accouch., Liège, ii, 41, 1881.
[7] Arch. de Tocol., xviii, 617, 1891.
[8] Med. News, lxx, 302, 1897.
[9] Lucina, Bologna, iv, 167, 1899.
[10] Brit. Med. Jour., ii, 1904, p. 1567.
[11] Glasgow Med. Jour., xvi, 25, 1881.

Had Dougall, however, tried simple opening of the sac of the spina bifida or of the spinal canal he would doubtless have failed to achieve his purpose, for it is necessary to pass a catheter up the canal into the cranium in order to reach the fluid.

The operation itself is quite simple. It requires only a knife and a catheter. A metal catheter is to be preferred, but a rubber one stiffened by a uterine sound inside it will do quite well (as was pointed out by Ira D. Stone).[12]

The indication for the operation is the birth of the breech of the child, and the retention of the head above the brim of the mother's pelvis, and the diagnosis or even the suspicion that the cause of delay is hydrocephalic enlargement of the fetal head. The presentation may have been the breech from the beginning of the labor, as in my second patient; or the shoulder may have presented and version been performed, as in my first case. If a spina bifida exist the opening in the spine may be utilized to pass the catheter up into the cranium (my second case); but if the opening prove too narrow (from flattening of the vertebræ) there would be no difficulty in making an opening higher up. If the hydrocephalus be accompanied by no spinal aperture, then a short transverse incision is to be made over the interspace between two of the dorsal vertebræ, and the catheter passed up the spinal canal. A long metal catheter will be needed unless the opening be made high up in the dorsal region, and that is not always practicable on account of the level in the pelvis at which the hydrocephalic head lies. The bend on the catheter should be slight, and the trunk of the child should be so held as to adapt the curve of the spine to that of the instrument as it passes up.

The advantages of the operation are manifest. There is first the smaller number of instruments required—a knife and a catheter. Second, there is the advantage of operating upon parts which are external to the passages and visible to the eye. There is, third, the rapidity with which the obstruction to delivery can be overcome in a case in which the further loss of time may mean uterine rupture. There is, fourth and most important, the avoidance of further manipulations in the vagina and uterus in a case in which, as a rule, there has already been much interference, instrumental and manual,

[12] Loc. cit.

and therefore much risk of septic infection. The object of the operation is to overcome serious obstruction to delivery existing above the brim of the pelvis by a simple operation performed outside the vulva; spinal paracentesis admirably meets this requirement. The alternatives are prolonged traction upon the fetal trunk with risks of uterine rupture, and tapping or perforating the hydrocephalic head by means of an instrument passed up alongside of the fetal body in the vagina and introduced into the uterus and then into the head as it lies above the brim. In both the alternatives the risks of rupture and of septic infection are very considerable.

With a rigid catheter I do not think that the operation ever ought to be unsuccessful. I have, however, a letter from a former pupil describing how in an otherwise suitable case the attempt to reach the intracranial fluid with a short catheter failed, and tapping the head itself had to be done. In that instance it is possible that the passage of a uterine sound up into the cranium might have succeeded. Another possible cause of failure may be the displacement of the vertebral bodies produced by prolonged and violent traction upon the fetal trunk, and the consequent difficulty in getting the catheter into the cranium. The obvious means of preventing this difficulty is the avoidance of prolonged and useless traction upon the fetal trunk in all such cases.

Case III.—On September 2, 1905, a woman, 22 years of age, and a primipara, was admitted to the Royal Maternity Hospital, Edinburgh; she had had two eclamptic seizures on the preceding evening. She last menstruated on Christmas Day, 1904. She was then living in Hong Kong. Her health then became poor; she suffered from headaches, and it was noticed that her face was puffy in the mornings. Her urine was scanty, dark brown in color; micturition was so painful that she dreaded passing water. These symptoms have persisted until the present time. She left Hong Kong in April, 1905, and reached London in June, after a rough passage during which she suffered both from morning-sickness and from sea-sickness. She made no attempt to diet herself. She had recently come to Edinburgh. Quickening had occurred at the sixth month, and she was now about one month short of the full term. The headaches, always occipital in position, had been very troublesome, and the swelling of the face had become more marked.

A week before admission all the patient's symptoms became more severe: the headaches were so bad that she could not sit down on account of them; the edema spread to the whole body, and she was passing very little water (a breakfast-cupful in 24 hours). She was first seen by a medical man on September 1; he put her on milk diet at once. The same evening, when she was talking to her mother, she suddenly began to twitch all over; the movements lasted for a few minutes, and were followed by unconsciousness. Her tongue was bitten. A few minutes afterward she had another fit. Half a grain of morphin was given hypodermically; she had no further fits during the night, and she was sent into the hospital on the following morning.

There was edema of the face and also of the limbs. The pulse was small, hard, and beating at the rate of 80. The urine became quite solid on boiling, and there were 7 grains of urea to the ounce. The patient was conscious and complained much of occipital headache. The abdomen had the appearances of a pregnancy at the seventh or eighth month. No fetal heart could be heard, but the patient thought that she felt movements. The fetal back was felt lying to the front and left side of the abdomen. I took occasion to examine the woman soon after her admission, and I called the resident surgeon's attention to the fact that the part at the brim which had been regarded as the fetal head was too soft for that structure and was too large for the breech; this observation is of importance in connection with the state of the fetal head found at birth twelve days later.

The patient was put to bed between blankets and was surrounded with hot bottles. Nothing but milk was given. The bowels were got to act freely with the help of compound jalap powder. Improvement began and continued for a week, the quantity of urine steadily increasing and the headaches becoming less severe; then, however, the headache again got worse and was practically constant, and the eyesight became dim. The ophthalmoscopic examination showed no distinct optic neuritis; but there was slight swelling of the right disc at the apparent lower and nasal quadrant, and the vessels were somewhat dilated. The pulse tension was high. There were slight labor pains. The amount of albumin in the urine increased to 1 grain per ounce, and the urea was estimated at 7 grains

per ounce. The skin and bowels were kept acting freely and the grave symptoms again passed off.

From September 9 to 13, the patient remained in a fairly satisfactory state. Then labor pains came on. At 4 A.M. on September 14, the os uteri was found to be three-quarters dilated, and through it a large soft presenting part could be felt; further examination disclosed bones with membranous intervals in the part palpated at the os. The diagnosis of hydrocephalus was thus made, and the obscurity of the antenatal diagnosis of the fetal presentation was thus cleared up. Labor went on satisfactorily, and the vault of the hydrocephalic head was born as a long sausage-shaped mass. As the skin of the scalp was seen to be peeling off in places, it was evident that the fetus had been dead for some days, perhaps since the supervention of the fits twelve days previously. As there threatened to be some delay in the birth of the base of the cranium, one of the resident surgeons (Dr. H. J. Dunbar) applied the forceps and with no great difficulty brought down the head. The infant, a female, showed macerative changes; the head was typically hydrocephalic, but there were no other malformations. The placenta showed one extensive hemorrhage situated centrally.

The patient recovered well from the delivery. No fits occurred at labor or in the puerperium, and the symptoms which had been so troublesome before delivery disappeared. Immediately before labor the albumin in the urine had been 1 grain per ounce and the urea 7 grains per ounce, and there had been hyaline casts present; on September 15 (the day after delivery) the urine had a specific gravity of 1030, the urea was the same (7 grains per ounce) and the albumin was 0.86 per ounce. On September 20, the albumin had fallen to 0.12 grains per ounce, and the urea had risen to 8 grains per ounce. On September 25, the analysis was practically the same, and the specific gravity was 1015. Three days later there was no longer any albumin: the urine was clear and without deposit. The patient left the hospital two days later: there was no edema and she stated that she felt quite well.

Remarks.—In this case the occurrence of antenatal death and maceration of the fetus made it possible for excessive moulding to take place, and so the hydrocephalic head was able to pass with slight assistance through the birth-canal. In this respect, as well as in the

fact that forceps were used to extract the base of the cranium, the case was exceptional. As a general rule when a hydrocephalic fetus presents by the head, forceps are entirely contraindicated and tapping or aspiration of the head is the correct treatment. Delay in such instances is always accompanied by the risk of uterine rupture, and therefore aspiration is to be performed as soon as the diagnosis of hydrocephalus and consequent obstruction to delivery is made. The association of eclampsia is interesting, but possibly not more than accidental. On the other hand the death of the fetus may have been instrumental in stopping the fits, although the strict dieting and other methods of treatment no doubt had a good effect.

THE FEMALE PERINEUM AND ITS REPAIR

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There is probably no portion of the female anatomy more difficult to comprehend, and about which there hovers a greater variety of conflicting opinions, than the pelvic floor or diaphragm, known generally as the *perineum*. Correctly speaking, the perineum is a more comprehensive structure than the true pelvic diaphragm, which term should be restricted to but one constituent part thereof, namely, the levator ani muscle, as the anatomist Luschka [1] has clearly indicated.

Much has been done in recent years to correct the haziness of our knowledge concerning this structure, and our hearty thanks are due to Dickinson,[2] Hanks,[3] Sellheim,[4] Studdiford,[5] Byron Robinson,[6] Burtenshaw,[7] and others for their indefatigable labors in this intricate subject. As a result of their studious efforts we are in the position to-day more thoroughly to understand the functions of the perineum, including its action during parturition, and the serious consequences that follow laceration of certain of its constituent parts. In this brief resumé of the literature of the perineum and its restoration after injury in labor or from other traumatism it becomes most imperative, therefore, that a correct idea be entertained of the anatomy of the parts as seen in the light of these latter day investigations.

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[1] "Anatomie."
[2] Amer. Jour. Obst., Sep., 1889.
[3] "Anatomie."
[4] Beiträge zur Geburtsch u. Gynäk. V. 2, 1901.
[5] N. Y. Med. Jour., Apl. 12, 1902.
[6] Jour. Am. Med. Asso., Sep. 10, 17, 24, Oct. 1, 8, 15, 22, 1898.
[7] N. Y. Med. Jour., Jan'y 10, 1903.

ANATOMY OF THE PERINEUM

The pelvic floor or perineum is made up of:—

(*A*) Skin.

(*B*) Muscles (Figs. 1 and 2). (1) *Vulvovestibular group:*
- (*a*) Erector clitoridis.
- (*b*) Musc. bulbi cavernosi.
- (*c*) Constrictor vaginæ (*sphincter vaginæ; constrictor cunni*).

(2) Transversus perinæi.

(3) *Anal group:* (*a*) Levator ani.
- (*b*) Sphincter ani.
- (*c*) Coccygeus.
- (*d*) Pyriformis.

(*C*) Perineal fascia: (1) Superficial fascia, superficial layer.
- (2) Superficial fascia, deep layer.
- (3) Triangular ligament.

Hart[8] remarks that the pelvic floor may be considered as composed of two widely over-lapping valves; the anterior of these comprises the bladder, urethra, anterior vaginal wall, and retropubic fat, and the posterior includes the posterior vaginal wall, the perineum, and the rectal wall. These structures are united by tendinous insertions in the median line to form the perineal body or wedge. Externally this line of union is represented by the well-marked perineal raphe, which extends from the posterior vulvar commissure to the anus.

The perineal body in the nulliparous woman is a dense firm mass having an external measurement or length from the anus to the vulva of about three centimeters. Sellheim classes perinæi as short if they measure less than 2.5 cm. (1 inch), and as very short if they are less than 1.5 cm. ($\frac{5}{8}$ inch). A perineum having a length of 4 or 5 cm., as is noted in cases of perineal relaxation due to subcutaneous laceration of the muscular fibers, is unusually long and proportionately weak. The formula has been established, therefore, that "the efficiency of a perineum is inversely proportionate to its length and directly proportionate to its depth or extension from the skin surface inwardly."

[8] "Atlases of Pelvic Anatomy."

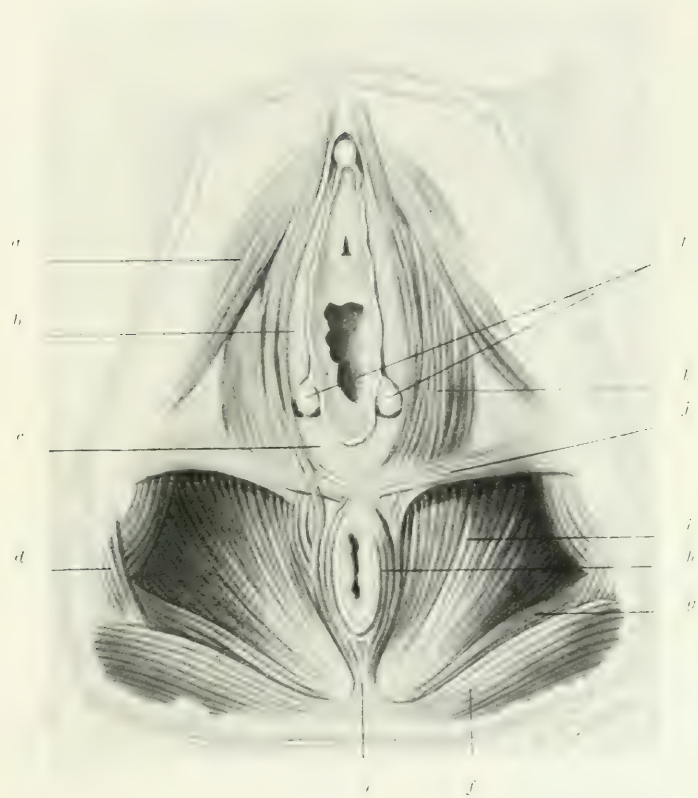

FIG. 1.—Muscles of the pelvic floor, as seen from above : *a*. ischiocavernosus; *b*. bulb of the vagina : *c*. fossa navicularis ; *d*. great sacrosciatic ligament ; *e*. coccyx ; *f*. gluteus maximus ; *g*. coccygeus ; *h*. external sphincter ani : *i*. levator ani : *j*. superior transperineal muscle ; *k*. sphincter vaginæ ; *l*. Bartholin's glands.

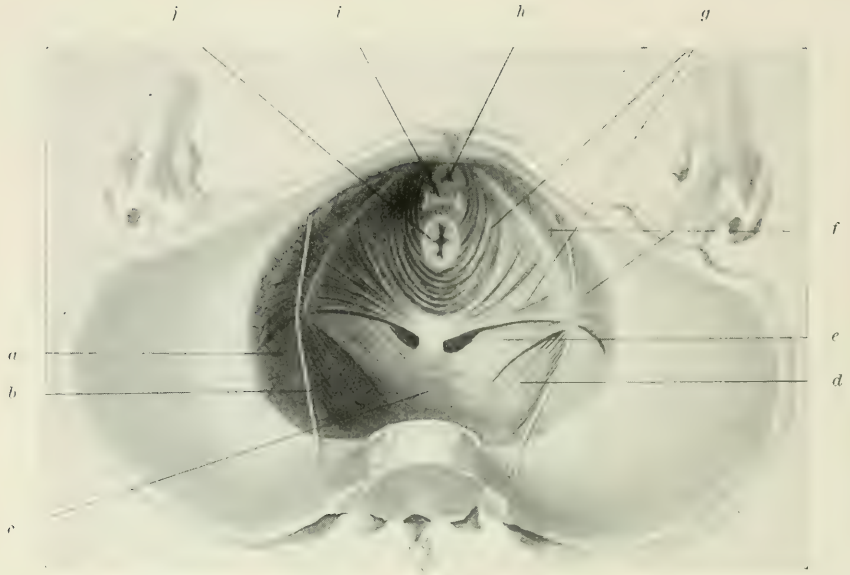

FIG. 2. The pelvic floor as seen from above: *a*. internal wall of the pelvis; *b*. obturator nerves; *c*. sacrum; *d*. pyriformis muscle; *e*. coccygeus muscle; *f*. obturator externus; *g*. levator ani; *h*. urethra; *i*. vagina; *j*. rectum.

FIG. 3.—Levator ani, as seen with the pelvis in a semi-recumbent position (modified from Dickinson).

Fig. 4.—Levator ani, as seen from the pelvic brim (modified from Dickinson).

Fig. 5.—Levator ani, as seen from below with the pelvis in an exaggerated dorsal position (modified from Dickinson).

FIG. 6.—Levator ani, as seen with the pelvis in the dorsal position (modified from Dickinson).

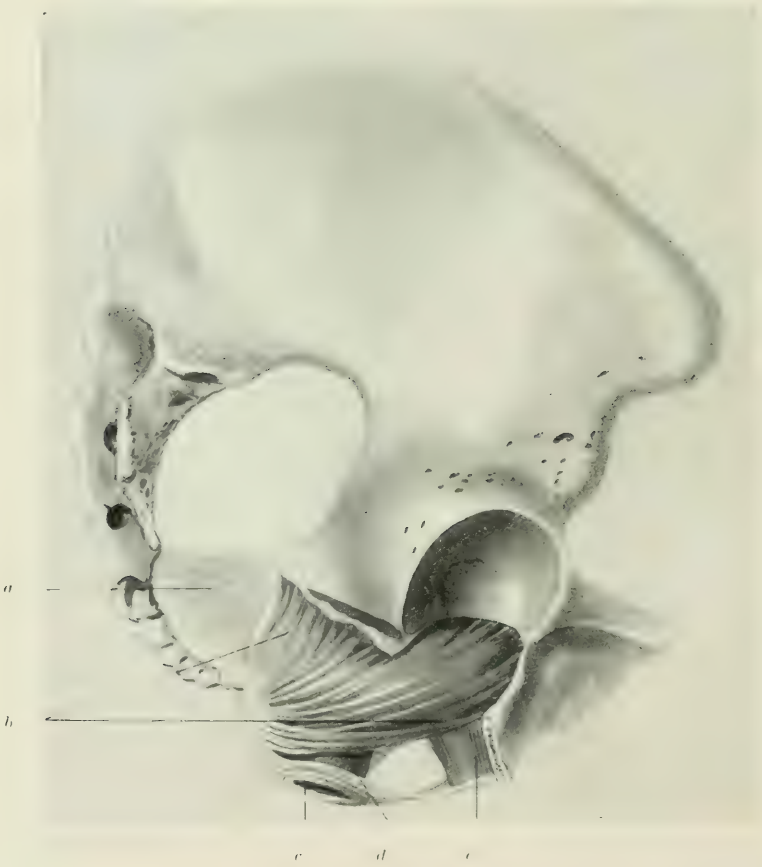

FIG. 7.—Side view of the levator ani muscle after removal of the ischium (redrawn after Luschka). *a*. Coccygeus; *b*. levator ani; *c*. anus; *d*. rectum; *e*. vagina.

FIG. 8.—External relation of the levator ani—the skin as if transparent.

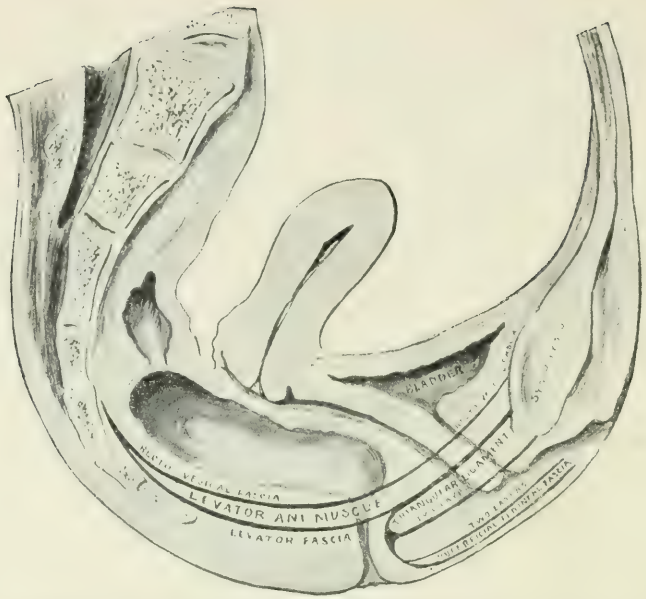

FIG. 9.—Diagram of the fascia of the pelvic floor in mesial section, to show how the levator is backed by "strong and dense" sheets of fibrous tissue. Five layers: 1, superficial perineal fascia, outer layer; 2, superficial perineal fascia, inner layer; 3, triangular ligament, outer layer; 4 triangular ligament, inner layer; 5, rectovesical fascia. (After Dickinson.)

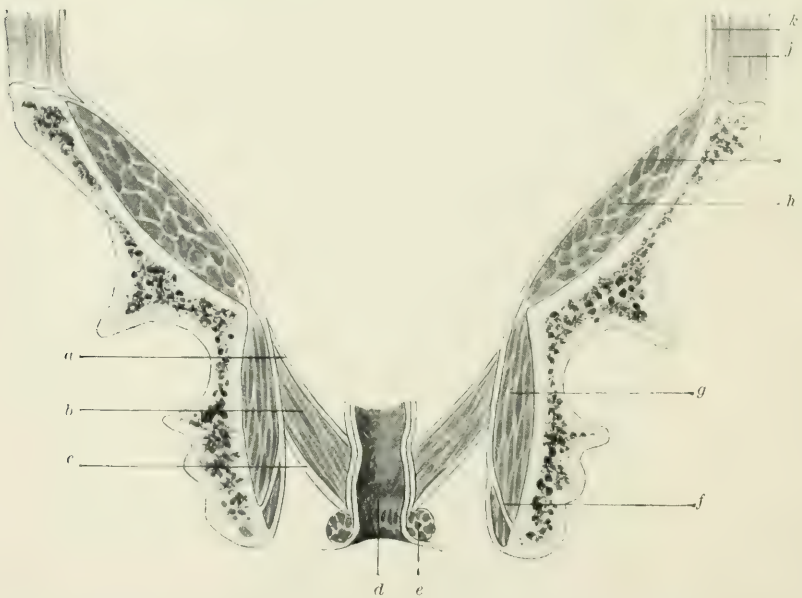

FIG. 10—Transverse mesial section of the pelvis, showing the fasciæ and muscles (redrawn rom Robinson Scholes). *a.* superior levator ani fascia; *b.* levator ani muscle; *c.* inferior levator ani fascia: *d.* rectum; *e.* external sphincter ani; *f.* Alcock's canal; *g.* internal obturator muscle; *h*, iliac muscle; *i.* iliac fascia: *j.* muscles of the abdominal wall; *k.* transversater fascia.

The functions of the perineum are constriction of the vagina, control of the anus, assistance in parturition, and the affording of support to the pelvic and abdominal viscera. These results are mainly affected through the agency of the great levator ani muscle, which is materially assisted by the transverse condensation of the perineal fascia known as the ischioperineal ligament of Savage, because of its attachment to the ischial tuberosities and the central perineal line. It is in reality the thickened lower border of the triangular ligament.

The Levator Ani Muscle.—Studdiford and Dickinson have within recent years, through a series of dissections and a study of frozen sections of the female perineum, arrived at a most accurate knowledge of the anatomy of this remarkable muscle, which is the chief supporting structure of the perineum. The former from his frozen sections describes the muscle as a "narrow V," the sides of which show a slight convexity toward the median line, while its opening is situated at the tip of the coccyx. Dickinson claims that "the muscle rather resembles a horse-shoe—a sling attached to the pubes in front, its sweep reaching horizontally backward to circle like a collar the rectum and vagina" (Figs. 3, 4, 5, 6, 7, and 8). In Studdiford's sections the vagina appeared as a crescentic opening and the rectum as a slit parallel to the long axis of the muscles. In the space between the vagina and the rectum Studdiford describes a remarkable structure which, he believes, plays an efficient rôle in the accomplishment of parturition. This is a "spindle-shaped band of tissue, distinct to the naked eye, lying between the two halves of the levator ani." It is about one-fourth of an inch in width in the median line and three-eighths of an inch wide where it unites with the levator. Upon microscopic examination this band of tissue was found to be composed "almost entirely of involuntary muscle, the fibers running in every direction and being closely attached to the levator ani." By the action of these fibers "the two portions of the levator ani are drawn close together, the posterior portion of the vagina is drawn upward under the pubes, and, with the halves of the levator approximated, the muscles are in a better position to draw the rectum and coccyx forward. These fibers also seem to be in close relation with the anterior end of the external sphincter, and tend to draw it nearer the pubes."

The levator is in reality composed of four paired muscles, designated occasionally as the ischiococcygeus, iliococcygeus, pubococcygeus, and the puborectalis or Holl's sphincter recti. These receive their names from the points of origin of their fibers.

As Luchka has indicated, the bony origin of the levator ani is principally from the horizontal ramus of the pubes on either side, 0.5 cm. ($\frac{1}{4}$ inch) below the upper border of the ramus, the fibers beginning 1.25 cm. ($\frac{1}{2}$ inch) from the median synphysial line and extending for about the width of two fingers. This, which is the densest portion of the levator muscle, extends nearly horizontally backward. A small band of the muscle fibers, not more than $\frac{1}{4}$ inch in width, rises from the inner surface of the ischial spine immediately anterior to the origin of the coccygeus muscle. Between these two points of bony origin is to be found the fascial origin of the muscle, which Dickinson describes as of a fine crescentic shape, the lowest portion of which is situated 5.5 cm. (2 inches) below the iliopectineal line. "This curved line of origin is intimately connected with the tissue of the pelvic fascia, so that the muscular fibers begin as tendinous bands which radiate out into the fascia and give much strength. Both on the upper and lower surface of the levator this fibrous tissue flattens and spreads out." There is, however, no constant connection between the muscle and the so-called arcus tendineus or "white line" of the pelvic fascia.

From these points of origin the muscle extends downward and backward, dividing into two unequal fleshy bundles, one of which extends anteriorly to the rectum and the other traverses its lateral and posterior aspects. According to Henle an intimate connection exists between the horizontal pubic fibers of the levator and the longitudinal muscular fibers of the vagina, whereby the latter can be materially shortened during labor. The anterior and smaller portion of the levator ani is thin and flat and crescentic in shape with its convexity below. Arising from the outer portion of the pubis it loops across the larger mass of muscle and rests in the rectovaginal septum.

The bulkier posterior portion of the muscle is composed of three distinct sections, the anterior and larger bundle closely hugging the rectum and connecting in large part with the fibers of the sphincter ani and also with the rectal walls, especially with the longitudinal

muscular fibers. The middle bundle, which is only 1 cm. long and 1 cm. broad, ends in an aponeurosis which is attached with its fellow of the opposite side to the tip of the coccyx. The smallest and posterior bundle is attached by means of a tendon to the anterior surface of the fourth coccygeal vertebra.

Dickinson has made some interesting observation concerning this muscle which may be briefly stated as follows: The levator measures from $\frac{1}{8}$ to $\frac{3}{8}$ of an inch (0.3 to 1 cm.) in thickness; it weighs one-quarter as much as the diaphragm and one-half as much as the external oblique muscle; it is capable of so firmly closing the lower end of the vagina that coitus and digital exploration are impossible; it is capable of exerting powerful resistance to the advancing head in labor, even totally obstructing the labor for a considerable period of time.

At this point it may be well in passing to note the stand taken by Browning[9] who claims that the levator ani is an unimportant structure of the body and is in fact a rudimentary muscle fading away with the passing of the caudal appendage. This view he asserts is substantiated by the weakness of the origin of the muscle, by the direction and insertion of its fibers, and by the well-grounded statements that muscles do not physiologically afford constant support to a part, and that the levator ani of the male is as well developed as is that of the female.

The Constrictor Vaginæ or Bulbocavernosus.—This muscle (Fig. 1) in large part fills in the gap between the anterior origins of the levator ani. It is a double muscle, thin and weak, each portion having its origin posteriorly in the perineal fascia at a point midway between the sphincter ani and the ischial tuberosity. A small fasciculus, according to Luschka, is joined to the sphincter ani. Converging anteriorly the muscle is inserted at three points, namely, on the inferior surface of the corpus cavernosum of the clitoris, on the posterior surface of the bulb, and into the mucosa between the clitoris and the urethral orifice. The main function of this muscle is to increase the turgidity of the parts by compressing the clitoridian veins; to a slight degree it may, as Lusk[10] has known, narrow the vaginal vestibule by forcing the turgid bulbs inward.

[9] Med. News, June 12, 1897.

[10] "Midwifery," 1886.

The Coccygeus.—This muscle (Figs. 1 and 2) has its origin on the ischial spine and lesser sacrosciatic ligament and extends in a fan-shape to be inserted into the tip of the coccyx and along the lateral aspect of the two inferior sacral vertebræ; it closes the small space back of the levator ani. It forms a flat musculo-tendinous plane with its anterior border in contact with the posterior border of the levator ani muscle. The posterior border bounds the anterior margin of the great sacrosciatic foramen.

The Sphincter Ani.—This muscle (Fig. 1), which is not a circular structure, as is generally supposed, but consists of two elliptical halves, has its origin posteriorly in the coccyx and the adjacent skin and superficial fascia. Its fibers after encircling the anus are inserted into the skin in front, into the central tendon of the perineum, and into the other muscles. (See under *Complete Lacerations.*)

The Pyriformis Muscle.—Taking its origin from the lateral portions of the second, third and fourth sacral vertebræ, from the inferior border of the great sacrosciatic notch, and from the great sacrosciatic ligament, the pyriformis muscle (Fig. 2) traverses the great sacrosciatic foramen and is inserted into the upper border of the great trochanter. It is covered by the pyriformis fascia and affords a bed upon which the sacral nerves rest.

The Erector Clitoridis or Ischiocavernosus.—This muscle (Fig. 1) arises from the inner surface of the tuberosity and ramus of the ischium, from the pubic ramus, and from the adjacent root of the crus clitoridis, and is inserted into the surface of the tunica albuginea of the corpus cavernosum near the point of attachment of the suspensory ligament.

Transversus Perinæi.—Existing in two portions, a superficial and a deep, the transverse perineal muscle contributes materially to the support of the perineum. It arises superficially from the inner surface of the ischial tuberosity and ramus together with the erector clitoridis, and extends inward and forward to be inserted into the tendinous center of the perineum. The *transversus perinæi profundus* of Henle is closely connected with the triangular ligament. It has its origin on the inner surface of the ischiopubic ramus and, spanning the subpubic angle transversely, both anteriorly and posteriorly to the membranous urethra and to the vagina, is inserted

partly in the median line and in part on the opposite pubic ramus. On account of its distribution around the membranous urethra, this portion of the muscle is designated as the compressor urethræ. Its fibers are comparatively thin and weak and are in great part unstriped. At the central point of union they interdigitate or blend with the fibers of the levator loop in the posterior vulvar region (Marcy).[11] It is the function of the transversus perinæi to keep the pelvic fascia tense and to assist in the approximation of the vaginal walls.

The Perineal Fascia.—The most important perineal structure next to the levator muscle is the fascia, a dense complicated tissue interlacing with and binding together all the other tissues. Robinson states that "the fascia of the pelvis consists of many distinctly defined layers which can be cleaved from each other," and this has been amply demonstrated to be true by careful pelvic dissections. Upon the restoration of the fasciæ depends the success of a plastic perineal operation.

As Brandenburg[12] has succinctly indicated, the pelvic fascia is attached laterally to the brim of the pelvis, anteriorly to the lower portion of the symphysis pubis, posteriorly to the spine of the ischium and to a tendinous band, the "white line" (arcus tendineus) joining these points. This line extends from the posterior surface of the pubis to the spine of the ischium. At the "white line" the pelvic fascia divides into the rectovesical or levator ani fascia and the obturator fascia or that covering the obturator internus muscle. The fascia covering the coccygeal and pyriformis muscles receives its name from these muscles, while that covering the sacrum is known as the sacral fascia.

The obturator fascia, divided into a superior and an inferior portion, is attached laterally to the iliac portion of the pelvic brim and to the upper border of the obturator membrane. Anteriorly it is attached to the pubic body and to the upper margin of the obturator foramen; posteriorly to the anterior surface of the great sacrosciatic ligament and to the anterior margin of the great sacrosciatic notch; and inferiorly to the margin of the obturator foramen, the descending pubic ramus, and the ascending ramus of the ischium.

[11] Jour. Am. Med. Assn., Oct. 21, 1899.

[12] Med. Record, Oct. 20, 1894.

The Levator Ani Fascia (Figs. 9 and 10).—Dickinson concisely gives the formation of the levator fascia as follows: The iliac fascia splits into three parts. One layer covers the inner surface of the obturator internus; a second very dense fascial sheet lines the levator ani on its under surface, and is known as the *levator, anal,* or *ischiorectal fascia* (*levator ani fascia inferior*—Robinson); the third division covers the levator on its inner or upper surface, and is the *rectovesical fascia* (*levator ani fascia superior*—Robinson).

The *rectovesical fascia* takes its origin from the "white line," and, forming the pelvic floor, is inserted in the median raphe behind the rectum, and in the rectal, vaginal, and vesical walls. It is very strong and short anteriorly, and is attached to the pubic bone above the obturator fascia. It forms the anterior and lateral true ligaments of the bladder.

The thin, transparent *pyriformis fascia* is a continuation of the obturator fascia above and of the coccygeus fascia in front. It is attached to the sacrum and to the upper margin of the great sacrosciatic foramen from the ischial spine to the sacroiliac point. It covers an area of about five square inches on each side of the pelvis.

The *coccygeus fascia* is a thin fibrous membrane covering the coccygeus muscle, and having an area of about 3½ square inches on each side of the median line of the pelvis (Robinson).

The *sacral fascia* is the thin fibrous structure which covers the inner surface of the sacrum and, in conjunction with the other fasciæ described, completes the internal lining of the pelvis and closes the pelvic outlet above the muscular layer of the floor.

The *ischiorectal* or true *levator fascia* (*levator ani fascia inferior*—Robinson) closely hugs the under surface of the levator ani muscle. It takes its origin in the "white line" and is dissipated upon the rectal wall. It is the most important structure in the operation of perineoplasty, since upon its restoration to its normal relations will largely depend the successful issue of the operation. Anterior to it is the triangular ligament.

The Triangular Ligament.—The *triangular* or *perineal ligament,* also known as *Camper's ligament,* is formed by the union of the obturator fascia of either side in the median pelvic line. It largely fills the gap between the pubic insertions of the levator ani muscle and is the main anterior means of fixation of the vagina,

whose walls it embraces. It is triangular in shape with lateral attachments to the pubic and ischial rami. It joins with the ischio-perineal ligament and levator ani muscle in giving the main support to the pelvic floor. It is continuous above and below with the levator fascia.

Perineal Laceration.—From the foregoing resumé of the perineal anatomy the disastrous results following the laceration of these main supporting structures may be readily comprehended. Solution of the continuity of the perineal muscles and fasciæ generally results from parturition, although traumatism from other causes, as from falls or during coitus may be noted. Thus, Royster [13] reports a complete perineal laceration produced in an infant at birth, the obstetrician mistaking the vagina for the mouth and exerting traction on the perineum with his finger introduced into that canal. Cases of central rupture of the perineum are exceedingly rare, statistics showing but one case in about 2000 labors. Still more infrequent is central rupture with the emergence of the head through the vulva, the rupture giving exit to a prolapsed arm. Such cases have been recorded by Charpentier, Kroner, Joffrion, Breiter, and Pujebet and Stérin.[14] This condition will be best repaired by converting the opening into a complete vaginoperineal laceration, in order to secure exact coaptation of the edges and the prevention of a fistulous tract.

Coles,[15] of Philadelphia, claims, as a result of careful examination after every labor, that 75 per cent. of primiparæ and 25 per cent. of multiparæ suffer some degree of perineal laceration, necessitating a suturation of the parts. As commonly encountered these perineal tears may be grouped into four distinct classes:

(1) *Partial lacerations,* in which the separation occurs in the median line of the perineal body from the posterior vulvar commissure for any extent down to or into the outer fibers of the sphincter ani.

(2) *Complete laceration,* the separation occurring in the median line of the perineal body through the sphincter ani and up the recto-vaginal septum as far, at least, as the upper margin of the internal

[13] Amer. Jour. Obst., March, 1902.
[14] Jour. des. Sci. Méd. de Lille, Oct. 12, 1901.
[15] Clinical Review, May, 1904.

sphincter. The ends of the sphincter are separated by an interval of a centimeter or more.

(3) *Subcutaneous* and *submucous lacerations* of the perineal muscles and fasciæ, the separation occurring in the line of the lateral vaginal sulci, and involving the bellies of the levator ani and transversus perinæi muscles.

(4) *Complete sulcal lacerations,* the traumatism being the same as in the preceding class with the addition of a tear involving the mucous membrane of the vagina (Fig. 11).

Tears of the third and fourth classes destroy the supporting power of the perineum and give rise to visceral displacement, as evinced by the speedy development of vaginal and uterine prolapse. Tears of the second class are immediately followed by gaseous and fecal incontinence.

THE RESTORATION OF THE PERINEUM

The operative indication is to restore the function of the injured tissues. As Robinson has shown, this includes: (1) The restoration of the levator ani fascia, superior and inferior; (2) the reunion of the fibers of the levator ani muscle so that it will functionate; (3) the restoration of the transverse perineal muscles, which draw the vagina lateralward, causing it to remain patulous; (4) the restoration of the ischioperineal ligaments; (5) the restoration of the rectovaginal septum and the posterior vaginal curve; and (6) the reunion of the edges of the sphincter ani in order to restore sphincteric action. These indications do not exist in their entirety in every instance. According to the extent and situation of the tear will depend the operative procedure that will be indicated. Thus, if the separation occur only in the median perineal line without involvement of the vaginal structures, a simple *perineorrhaphy* must be performed, and this is termed *primary* if done within twenty-four hours of the time of injury. If in addition the vaginal tissues are involved in the laceration the operative procedure is termed a *kolpoperineorrhaphy,* which may be *primary* or *secondary,* the latter being more correctly known as a *perineoplasty.*

PRIMARY OR IMMEDIATE PERINEORRHAPHY

Unless there be some contrary indication the immediate repair of a laceration of the perineum is the proper course to pursue. This

FIG. 11.—Relaxed perineum, showing a gaping vulvar orifice.

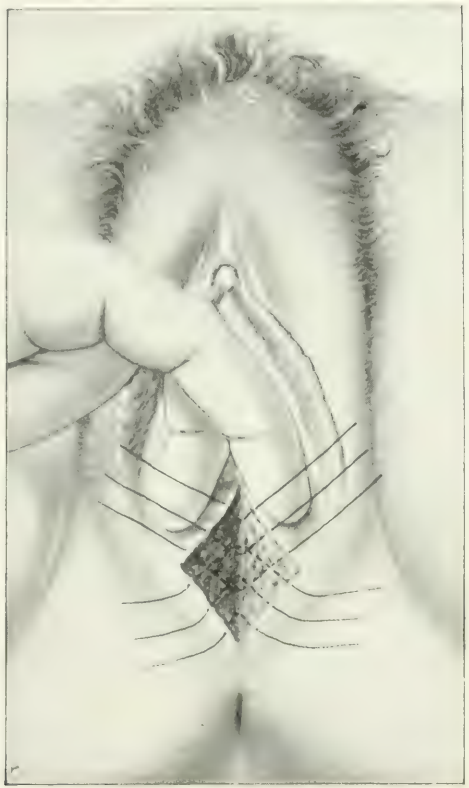

FIG. 12.—Simple median tear; the vaginal and perineal sutures are shown.

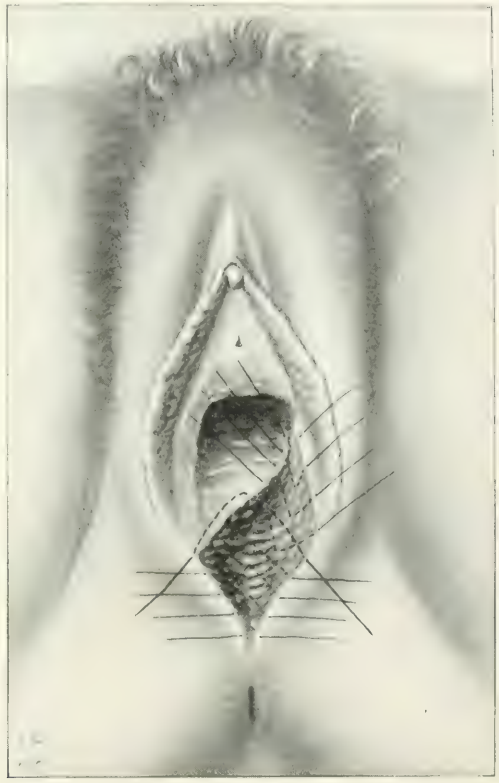

FIG. 13.—Combined median and sulcal tear; the sulcal, perineal, and crown sutures are shown.

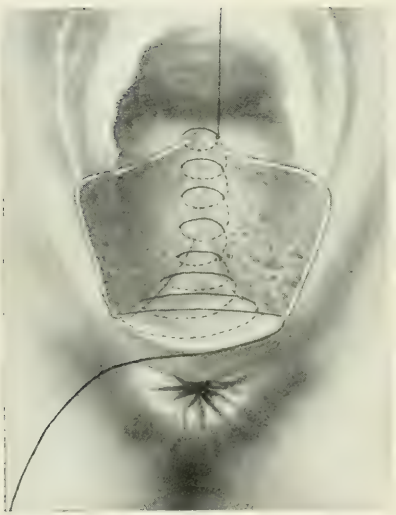

FIG. 14.—Martin's operation ; first stage of the continuous suture.

FIG. 15.—Martin's operation ; suggesting the final stage of suturation.

operation requires no anesthesia, as a rule, other than the use of an 8 per cent. solution of cocain injected around the margins of the wound. The number and situation of the sutures will depend upon the site and extent of the injury. In the greater number of the simple median tears without much involvement of the posterior vaginal wall, three or four interrupted sutures of silkworm gut, deeply inserted on the perineal aspect at ½-inch intervals and buried to the depth of the tear, will be effective. As Mabbott [16] has shown in order to secure proper coaptation each suture should be so introduced as to be elliptical, with its long axis transverse. An outward sweep of the needle to each side of the wound includes sufficient tissue to give the necessary support. These sutures may be removed in from eight to ten days.

When the tear extends some distance up the median line of the vaginal wall it becomes necessary to introduce vaginal sutures, which are carried down into the perineal structures in a similar manner as the perineal sutures, but at a right angle to these (Figs. 12 and 13). For this purpose the stronger chromicized catgut makes the best suture material; it holds the edges in close apposition, and, if the 20-day variety be used, the absorption will not occur before firm union has taken place. A. Martin uses a continuous buried catgut suture instead of the interrupted transverse sutures in closing these median tears; this insures a firmer cicatrix and obviates the necessity of subsequently removing the sutures (Figs. 14 and 15). A tear extending up one or both sulci requires the same suturation as in Emmet's secondary kolpoperineoplasty.

PERINEOPLASTY AND KOLPOPERINEOPLASTY

The late operation upon the pelvic floor is closely identified with many names that are famous in gynecology, preëminent among which may be mentioned those of Hegar, Emmet, Tait, Kaltenbach, Sims and Simon. By far the most important process in the late operation is the coaptation of the edges of the divided structures. In so far as the muscular fibers have not atrophied from disuse the reunion of their edges will restore in large part the normal tenseness and rigidity of the perineum. It will be seen, therefore, that in order to secure the greatest degree of efficiency there must be a

[16] Med. Record, Apl. 4, 1903.

minimum sacrifice of tissue and as early an operation as is feasible. Numerous are the methods and modifications that have been suggested for this purpose, but these all classify themselves into three distinct groups as follows: (1) Those in which the denudation and operative procedures are confined altogether to the perineal body—*perineoplasty*. These methods include, primarily, all the older and now almost obsolete operations in which there is made a denudation merely of the mucous membrane with apposition of the raw surfaces until healing occurs, but without any attempt to restore the perineal body to its original form, as in the Baker Brown method; and, secondarily, the more recent flap-splitting methods of Tait and others, in which no tissue is removed, but mucous flaps are made, pushed forward and backward, and the sides of the vulva and denuded area brought together by transverse sutures, thereby forming a longitudinal instead of a transverse cicatrix. Such operations are cosmetic merely and do not restore the original condition of the parts. (2) Those in which the posterior vaginal wall is included in the denudation and the deeper structures are drawn together without any special attempt at reunion of the divided fibers, as in Hegar's operation—*median kolpoperineoplasty*. (3) Those in which the vaginal sulci are opened in addition and the retracted muscles are brought into their normal relationships, as in Emmet's operation—*lateral kolpoperineoplasty*. This group includes those operations which are most rational in their design and most effective in their results.

PERINEOPLASTY

The older methods of perineal operation, including those of Baker Brown, Sims, Simon and others, were but the pioneers of the finished operation of to-day. Their objects seemed to have been the diminution of the vulvar outlet and the formation of a long but shallow perineum. Such methods paid no attention whatever to the deeper muscular and fascial tissues; the denuded area encircled the lower margin of the vulvar cleft and but slightly encroached upon the vaginal tissues. The sutures were almost entirely introduced from the perineal surface and were shotted directly in the median line, or secured by means of the double vertical metal or hard rubber bar. Modifications suggested in more recent years introduced in addition some median vaginal sutures, but these,

Fig. 16.—Tait's operation for incomplete laceration: the method of denuding.

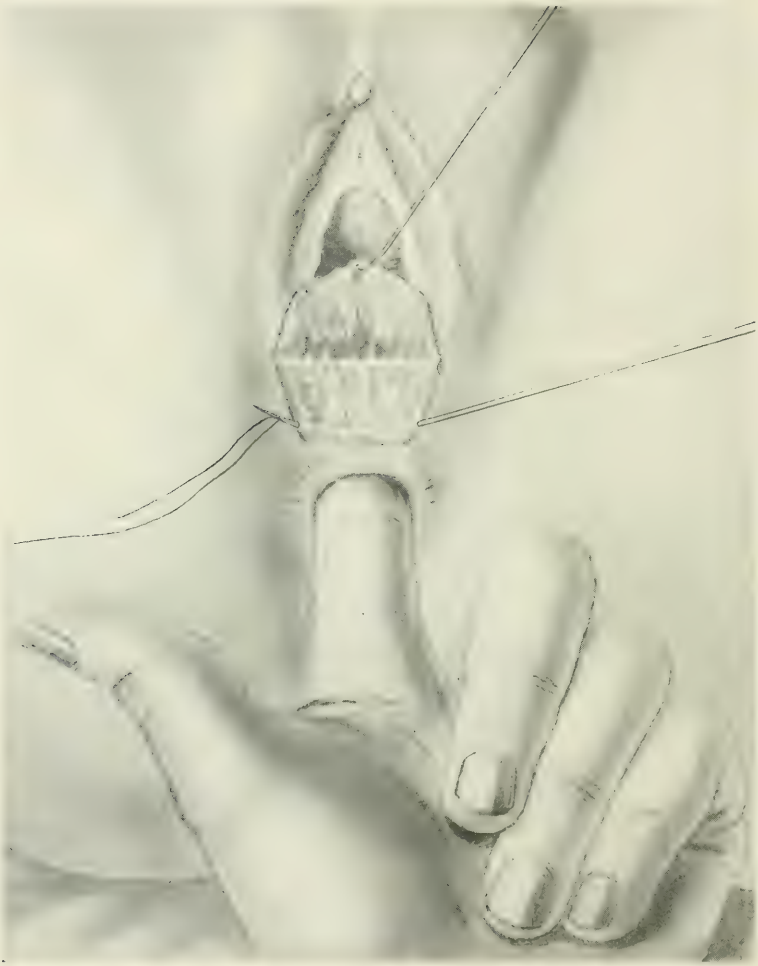

Fig. 17.—Tait's operation for incomplete laceration; method of passing the sutures.

unless deeply inserted, failed to grasp the divided and retracted levator and transverse perineal fibers. Thus, as Bantock [17] has indicated, the first important improvement on the primary crude approximation of the denuded edges was the triangular method of Dieffenbach, the sutures being inserted in the rectum, vagina, and perineum. Baker Brown divided the sphincter bilaterally, and Simon made extensive lateral denudations in order to secure more perfect results. Von Langenbeck dissected a tongue-shaped flap from the vaginal recess and used it to cover the median line of sutures.

FLAP-SPLITTING OPERATIONS

A considerable following still adheres to the flap-splitting operation of Lawson Tait and its modifications, notwithstanding the fact that this method almost absolutely ignores the anatomic relations of the parts. Von Ott [18] states that the claim that no tissue is sacrificed in the flap method is only an apparent advantage; there is in reality no economy of tissue material. The flap is thicker than the tissue removed in denuding, and it includes a certain amount of muscular structure, while a considerable portion of the flap lies in a loose fold in the vagina and is, accordingly, lost to the new perineum. Sloughs are not infrequent, and greater tension is required to maintain contact of the apposed surfaces owing to the material lost in the flap. The points in favor of the method are simplicity and rapidity of execution.

The operation, as devised by Tait (Fig. 16), consists in splitting the rectovaginal septum along the line where the posterior vaginal wall meets the anterior rectal wall; a flap consisting of the posterior vaginal wall is then turned up, and a flap composed of the rectal wall is turned down, thus making a separation of the rectum from the vagina. The tissues are then brought together laterally between the separated flaps so as to form a new perineal body (Fig. 17). The line of incision is indicated by a distinct white line which marks the union of the margin of the original tear, and which is joined at either end by two well-marked white lines not quite at right angles to the main line, but as Tait [19] says "touching it like

[17] Text-book of Gynecology.

[18] Arch. f. Gyn., April, 1891.

[19] Brit. Gyn. Jour., Aug. 1891.

the sides of an isosceles triangle whose apex would be situated a little distance in front of the arch of the pubis." The sutures are to be placed deeply and in such a manner as to disinter and bring into close apposition the injured muscular fibers. Spanton [20] and Duke [21] have made slight modifications of the flap-splitting operation. Duke introduces his sutures from without and makes his incision deeper than in the Tait operation.

MEDIAN KOLPOPERINEOPLASTY

Hegar [22] may be regarded as the pioneer in the deeper invasion of the vaginal tissues in the effort to restore the perineal body. He made a triangular denudation extending up the posterior vaginal wall and laterally as far as the tear extended (Fig. 18). This denudation removed the mucous membrane and cicatrical tissue only and did not include an exposure of the deeper muscular fibers. The apex of the triangle reached to the crest of the rectocele, and below the width of the triangle was limited by the myrtiform caruncles. The sutures were introduced from the vagina and from the external perineal surface (Fig. 19). The apex and upper portion of the triangle were closed by a continuous catgut suture tied above and drawing together the vaginal mucosa as far down as it could be approximated without undue tension of the vaginal walls. This gave a firm support for the rectocele, and resulted in a narrowed and shortened posterior vaginal wall. The lower portion of the tear was closed by three or four silkworm-gut or silver sutures introduced deeply from the perineum and swept around in a large curve through the structures of the perineal body. When tied these sutures formed a vertical line upon the external perineum and closely approximated the labia majora, the upper stitch about corresponding to the site of the myrtiform caruncles.

Following this suggestion of Hegar, other operators abroad and in this country have made trifling modifications of the posterior median operation. They are all deficient, however, in that they fail to make a restoration of the injured structures, and only indi-

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[20] Provincial Med. Jour., July 1, 1895.

[21] Montreal Med. Jour., March, 1891.

[22] Gyn. Opérat., French Edition.

Fig. 18.—Hegar's operation: area of denudation.

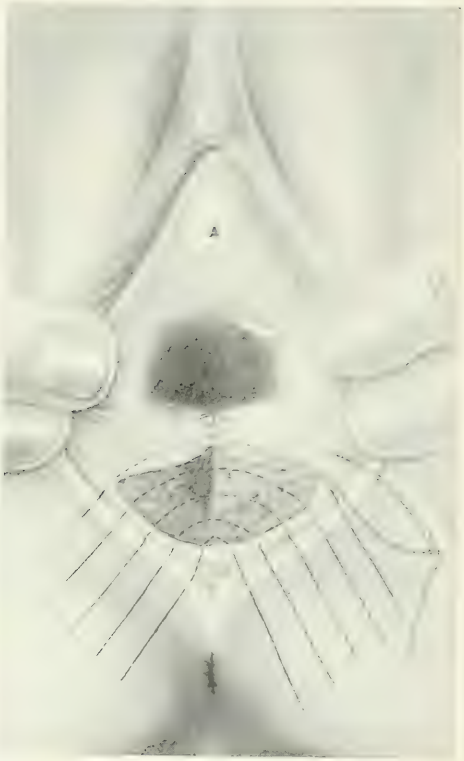

Fig. 19.—Hegar's operation. Method of passing the perineal sutures and the vaginal running suture.

FIG. 20.—Emmet's operation, showing (*s*) sulcal sutures; (*p*) perineal sutures; (*c*) crown suture.

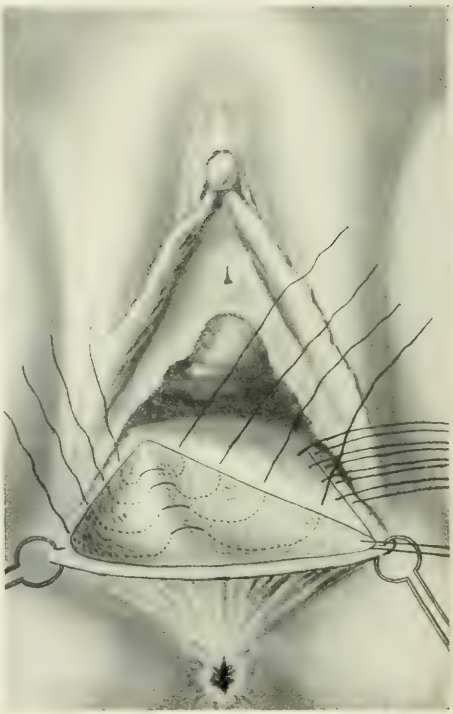

FIG. 21.—Emmet's operation, showing the method of inserting the sulcal sutures by carrying the median tongue to the opposite side.

rectly and incompletely approximate the gaping muscles. They do not, therefore, give the proper support to the vagina and pelvic organs.

LATERAL KOLPOPERINEOPLASTY

It remained for the distinguished Thomas Addis Emmet, of New York, to solve the problem of perineal repair by the application of appropriate surgical procedures to an intimate anatomic knowledge of the parts. Realizing that an injury can be repaired only by surgical measures directed toward the injured structures, he concluded that the torn levator muscle and fascia must be reunited in order to restore the normal pelvic support. His work was anticipated to a degree by Bischoff who spared the median vaginal column and made his denudation high up on either side; below he made a posterior median denudation with lateral butterfly wings extending up the lower third of the vulvar cleft. Emmet [23] determined the extent of the new posterior vaginal wall by fixing in the apex of the rectocele a tenaculum and drawing it forward to meet the approximated myrtiform caruncles. This formed a triangular-shape tongue to either side of which lay the vaginal sulcus in which is to be found the injured belly of the levator ani muscle. The three points, namely, the crest of the rectocele and the two myrtiform caruncles, form the guiding points for the denuding process, and through them is passed the "crown stitch," as will be seen directly. If the crest of the rectocele be carried to either myrtiform caruncle the opposite vaginal sulcus will be opened up, and the white line of cicatrix therein will indicate the extent to which the denudation must be carried. The entire anterior surface of the rectocele below the crest is stripped of its mucosa and the denudation is carried back and up into the triangular cavity of the sulcus upon either side. Emmet removed the mucosa in long strips by means of scissors curved on the flat, care being taken not to leave islets of the mucous membrane. The denuded area when complete resembles roughly a butterfly in shape, with a central rounded space and two triangular areas to the sides extending up the vaginal sulci. These spaces are closed by three sets of sutures, namely, the sulcal, the perineal, and the "crown suture" (Figs. 20 and 21).

The sulcal sutures are V-shaped beginning at the apex of the

[23] "Principles and Practise of Gynecology."

triangle of the sulcus and descending toward the perineal body at intervals of ¼ inch. They are directed from above downward and from behind forward and are introduced deeply so as to catch the retracted fibers of the torn levator. When tied they draw the perineal structures up into the vaginal sulci and thereby restore the normal relations of the parts.

The "crown stitch" includes the three guiding points of the denudation. Introduced at the left myrtiform caruncle, it is made to pass deeply through the tissues outward and then upward in a curved manner to emerge at the crest of the rectocele; it is immediately reintroduced at this point and with a corresponding sweep and curve traverses the tissues of the opposite side to emerge at the right myrtiform caruncle. When firmly tied it approximates the two myrtiform caruncles in the median perineal line and also draws down the triangular vaginal flap to form the posterior vaginal wall. It is the topmost stitch of the new perineum, leaving an elliptical gaping surface beneath. By the introduction of this stitch the vulvar orifice is diminished and the closed sulci pass out of view. Three or four perineal sutures are now inserted as already described in the primary operation for simple median tear of the perineum.

When these sutures are all introduced the vaginal wall lies in normal approximation, there is no protrusion of the mucosa through the vulvar cleft, and the labia majora are in contact as in a nulliparous woman.

This is now recognized as the ideal perineoplastic operation. There have from time to time been suggested slight improvements in the technic, but no radical change in the principles of the operation. Thus, Studdiford modifies the "crown suture" by introducing it at the upper margin of the external sphincter, which is then drawn forward and the needle reintroduced under the denuded tissues and passed out to or through the muscles of that side and then down to the point of entrance. C. P. Noble [24] passes the "crown stitch" superficially through mucous membrane only. Beneath it he passes two sutures through the vaginal mucous membrane and through the anterior border of the exposed levator muscle, the tissues in the median line in front of the rectum, and the corresponding tissues

[24] Am. Gyn. and Obst. Jour., April, 1897.

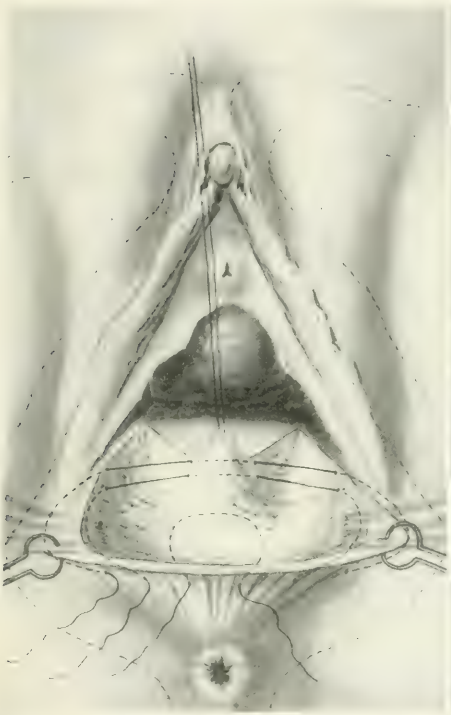

FIG. 22.—Hirst's modification of Emmet's operation, showing the closure of the sulci by through-and-through sutures.

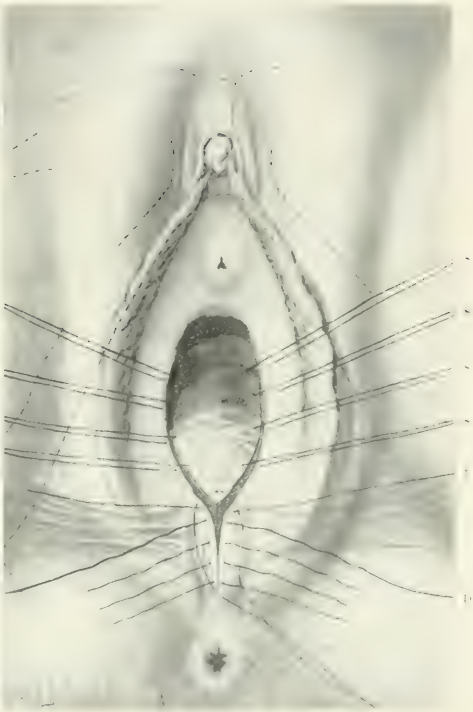

FIG. 23.—Hirst's modification of Emmet's operation, showing: (*s*) sulcal sutures, and (*p*) perineal suture, (*c*) crown suture, and (*f*) special fourchet suture.

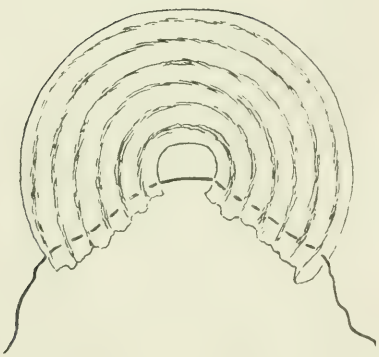

FIG. 24.—Diagram showing the course of the suture through the ends of the retracted sphincter muscle.

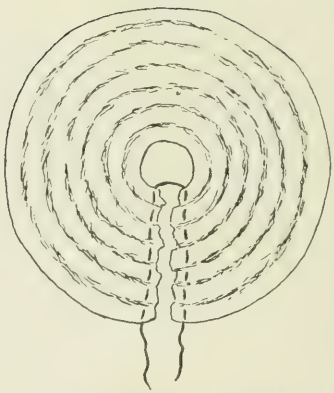

FIG. 25.—Diagram showing the ends of the sphincter muscle approximated when the suture is tied.

of the opposite side. A fourth suture is introduced at the plane of the hymen which catches the pelvic fascia and the transverse perineal muscles; it lies entirely below the plane of the levator muscle.

Hirst,[25] by his method of denuding in three triangular flaps, precludes the possibility of leaving islets of mucous membrane and shortens the operation by many minutes. He also closes the sulci by two or three sutures passed directly across the vagina, and introduces a special fourchet stitch above the "crown suture" to close the gaping vulvar orifice at this point (Figs. 22 and 23). Goldspohn[26] brings the levator muscle and fascia together by operating within the pelvic lumen and beneath the vaginal posterior and lateral walls. He does this by first detaching these walls for a distance of from six to eight centimeters in the median line and about half way up on the sides. The two opposing lateral portions of the deep pelvic fascia and levator ani are then by suture brought into direct contact in the median line in front of the rectum and beneath the vagina. The flap of posterior vaginal wall now lies spontaneously adjusted into longitudinal folds upon the bridge which has been constructed for it. This method differs from the Emmet operation in that in the latter method there is approximation but not actual contact of the deep lateral tissues.

Goldspohn's method is very similar in its results to that suggested by Harris,[27] who resects a portion of the puborectalis muscle so as to shorten the pelvic floor. He does this by carrying an incision up each lateral vaginal wall for from three to five centimeters, a little posterior to the median line; the vaginal wall is raised in a flap each way from the incision, and an incision is made through the perivaginal connective tissue parallel with the edge of the muscle, which is thereby exposed. The muscle is then dissected out ventrally as far as the symphysis and dorsally to its curve around the rectum, and a portion resected. The vaginal flap is then closed over the sutured muscle.

The methods recently suggested by Holden[28] and Somers[29]

[25] Univ. Med. Mag., June, 1898.
[26] Amer. Jour. Obst., June, 1901.
[27] Jour. Am. Med. Asso., Dec., 1899.
[28] Am. Jour. Obst., Oct., 1905.
[29] Jour. Am. Med. Asso., Nov. 11, 1905.

also aim at direct union of the divided muscle fibers. Thus, Holden, after denuding a triangular area, draws the levator muscle to the median line from either side and passes a traction-suture through it. These edges are drawn together and two external sutures of silkworm-gut are passed through skin, subcutaneous tissue, and levator muscle drawn forward to the median line. These sutures when tied closely approximate the muscles in the median line where they are united by figure-of-eight sutures and the traction-sutures removed, after which the denuded surface is closed with catgut. This operation brings the muscle together in front of the rectocele. This is similar to Reed's [30] operation save in the method of denuding, Reed splitting up a flap as in Tait's operation. Somers's " fishing operation " consists in making a triangular denudation as in the Hegar operation, after which the edges of the levator muscle are grasped by vulsellum forceps and drawn out from the surrounding tissues. The two muscles are then united by a running silkworm-gut suture, and the triangular area is closed in a similar manner.

W. Gill Wylie [31] made a nearly square-shaped denudation with its longer axis extending up the vagina, except in cases in which the rectocele was large, when the long axis extended from side to side. The sutures he inserted largely within the vulvar orifice, entirely closing the denuded area of the posterior vaginal wall over and in front of the rectocele, which is thereby reduced. A few perineal sutures were then introduced in the usual manner.

These modifications all contain valuable suggestions which may be put into operation in individual cases, but can scarcely be said to be applicable to all, save, possibly, Hirst's method of rapid triangular denudation. In no wise do they supplant, or attempt to supplant, the admirable and scientific work of Emmet.

COMPLETE PERINEAL LACERATION

Complete laceration of the perineal body through the sphincter ani into the rectum and up the rectovaginal septum for varying degrees involves an entirely different proposition from that which

[30] Jour. Am. Med. Asso., Aug. 30, 1902.
[31] Amer. Jour. Obst., July, 1894.

has thus far been under consideration. Such tears lie in or near the median plane of the pelvic floor and do not in the least result in levator tears with corresponding loss of support to the pelvic viscera. They do, however, introduce a new and very trying feature into the case. There is now present more or less loss of control over the lower bowel, so that the patient is subjected to the mortification and unpleasantness of gaseous and fecal incontinence. It is to be noted that even in cases in which the anus presents a normal appearance there may be a rupture of the sphincter with considerable separation of the ends.

Kelly's [32] division of complete laceration of the perineum is, therefore, an excellent one. He groups these tears as follows: (1) Those in which the tear extends barely through the sphincter. (2) Those in which the ends of the sphincter are separated by a well-defined interval of a centimeter or more. In the first of these groups the muscle is merely divided, the ends lying in close approximation, or separated only by a plug of scar tissue. The muscle no longer fully retains its sphincteric function. In the second group the condition is decidedly more aggravated so far as the external sphincter is concerned. In both groups, however, continence is more or less maintained by the increased activity of the internal sphincter. A third group of tears includes those which pass through the internal sphincter and up the rectovaginal septum.

Dickinson [33] makes the interesting statement that complete rupture of the perineum never takes place in the median line, but he admits that when these tears are examined some months after the injury they appear as if in the median line. The retracted sphincter ani can be outlined by means of two dimples in the buttocks where the ends of the sphincteric fibers rest. The lower margin of the anal orifice is formed by the concave retracted sphincter, while the upper margin consists of the torn and ragged rectovaginal septum which resembles an inverted V. He further states that the sphincter is not a circular muscle, but consists of two elliptical halves having the skin, superficial fascia and coccyx for its posterior origin, and its insertion in the skin anteriorly, in the central tendon of the perineum, and in the other muscles.

[32] Bull. of Johns Hopkins Hosp., Jan., Feb., Mar., 1899.

[33] Am. Gyn. and Obst. Jour., Vol. 6, No. 5.

PRIMARY COMPLETE PERINEORRHAPHY

The primary operation for complete perineal laceration is easy of performance when compared with an operation at a later period. It is, as Burrage [34] points out, possible at the time of injury to distinguish with more or less accuracy the individual muscles that have been torn and to reunite their ends. This may be accomplished by direct suturation of the vagina with silkworm-gut introduced from side to side, the sutures emerging at the edge of the rectal mucosa on one side and being reintroduced at the corresponding point on the opposite side. These vaginal sutures are carried down to the superficial sphincter stitch. The important sphincter sutures are introduced as follows: In order to catch the deepest sphincter fibers the first suture is introduced close to the original anal opening and behind the retracted muscle fibers. The needle is carried deeply through the tissues with a wide outward sweep and is caused to emerge at the depth of the torn perineal body. Here it is immediately reintroduced and carried with a similar outward sweep through the tissue to emerge at a point on the opposite side of the anal orifice corresponding to the primary point of insertion. The outermost retracted sphincteric fibers, which can be clearly seen in the fresh tear, are now caught in a second suture which is carried as a deep V through the depth of the perineal wound. When the first sphincteric suture is approximated and tied the anal orifice is closed. On approximating the ends of the second suture and tying them, the first sphincter suture is hidden from view, only the long free ends emerging from the puckered anal orifice. If these two sutures be carefully and properly introduced, sphincteric action will be restored and the operation will be a success. The usual transverse perineal sutures complete the process.

SECONDARY COMPLETE PERINEORRHAPHY

As in the case of the sulcal tears, Emmet's operation has proved the standby for the surgeon here. The same unfortunate condition of muscular atrophy as in the other form of secondary operation must be contended with in this case, and this militates against a thorough result in the operative procedure. There is probably no

[34] Bost. M. and S. Jour., Aug. 1, 1895.

minor gynecologic operation which requires greater care in the details of the technic. In the preparatory treatment we would most emphatically emphasize Burrage's suggestion of preliminary stretching of the sphincter. This overcomes the possibility of postoperative sphincteric irritability which may cause by spasmodic contraction a tearing apart of the approximated ends of the sphincter. Fritsch [35] adopts instead Simon's suggestion to divide the sphincter posterially, after, however, instead of before the operation. The tenotomy is done subcutaneously, and he claims that the sphincter regains its function perfectly.

In the process of denudation care must be taken to expose completely the retracted ends of the sphincter muscle. All superincumbent tissue must be excised. This can best be accomplished by seizing the umbilicated scar, already referred to, with a double tenaculum and after drawing it out snipping it off with scissors. A triangular denudation is then made on each side of the torn vulvar orifice, the apices meeting in the median line of the septum about three centimeters above the apex of the tear. This is the now famous "butterfly" denudation of Emmet. It should be made from below upward to avoid interruption from the flow of blood, and the work may be greatly facilitated by the introduction of a finger into the rectum to place the septum upon the stretch. It will be readily seen that when the lower and inner margins of the butterfly wings are approximated the anal orifice and the rectovaginal septum will be closed, while approximation of the outer and upper margins will close the perineal body and lower vaginal wall.

The sutures, which may be of silver wire or silkworm-gut—personally I prefer the latter—must be passed with great accuracy to insure close coaptation of the muscular edges. The first sphincteric stitch is of the greatest importance, in that it is intended to unite the sphincter edges and restore the anus. It is passed in the manner already described for the primary operation. It hugs the edges of the retracted sphincter on either side and secures its support at the angle of the tear in the septum. Its course is diagrammatically shown in Figs. 24 and 25. The second sphincter suture catches the exposed edge of the muscle which had been covered by the umbilicated scar and completes the sphincteric union. The

[35] Zent. f. Gyn., No. 2, 1901.

remaining four or five sutures are passed at equal intervals through the perineal body in the usual manner.

The old method of keeping the bowels constipated for eight or ten days is no longer employed. It is better to administer a saline, preferably Epsom salts, one dram every one-half hour until the bowels open slightly, beginning the day after the operation. This may be repeated daily in order to secure a gentle catharsis; it prevents the formation of scybala and obviates the danger of injury from these hardened masses.

Various modifications of the technic of this operation have been suggested from time to time. Thus, Hegar used for the internal sphincter fine silk and for the external sphincter fine catgut. Kelly insists upon the more thorough dissection of the ends of the sphincters and the excision of all fibrous tissue (See Figs. 26 and 27). He also separates the external sphincter from the overlying skin for a considerable distance, so that after the sutures have been introduced it will lie deeper and the danger of infection be lessened. After suturation of the rectal mucosa the sphincter sutures, previously inserted, are tied and a silkworm-gut suture is passed to remove the tension from the buried sphincter sutures. The internal sphincter, if divided, is sutured by fine silk threads introduced through the rectum. The method advocated by Leopold and Wehle [36] is practically the same as that recommended by Kelly. Baldy [37] states that he has frequently had to contend with infection of the rectal side of the wound, and to obviate this he suggests a high incision in the vagina and the turning down of a flap with suturing in such a manner as to do away with the rectal wound.

The direct suturation of the ends of the sphincter muscle was also advocated in 1895 by Le Conte,[38] of Philadelphia. He employed for this purpose two buried catgut sutures on either side, introduced in the same manner as a tendon suture. When the opposed sutures were tied the sphincter ends were tightly approximated. The "purse-string" suture of Percy Boulton,[39] which passes through the ends of the exposed sphincter ani and up the

[36] Geburts u. Gynäk., Bd. 2, Leipzig, 1895.
[37] Phila. Med. Jour., June 3, 1891.
[38] Am. Jour. Obst., June, 1895.
[39] Brit. Med. Jour., Dec. 20, 1890.

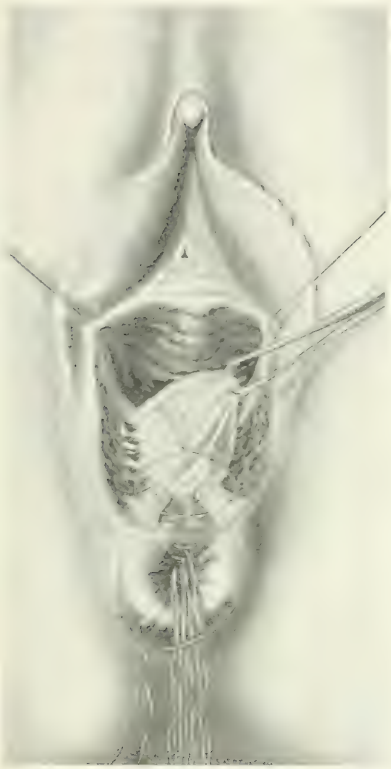

FIG. 26.—Kelly's operation for tear through the sphincter ani; showing the recto-vaginal septum united and the sphincter sutures inserted but not tied, and the vaginal flap drawn up (redrawn after Kelly).

FIG. 27.—Kelly's operation.: showing the sphincter suture tied (redrawn after Kelly).

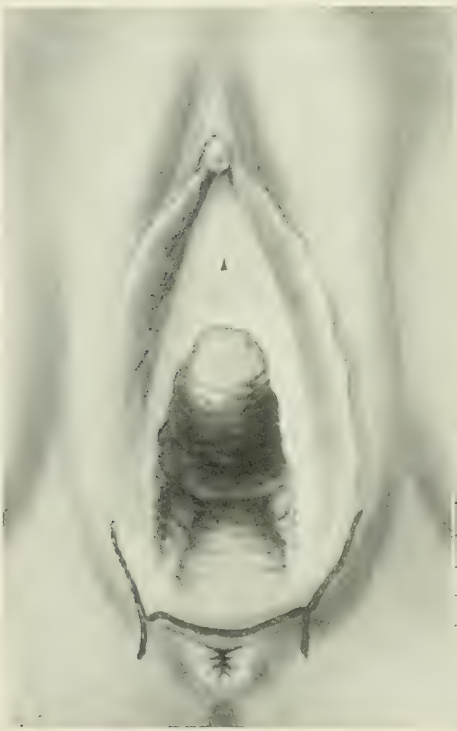

FIG. 28.—Tait's operation for complete laceration through the sphincter; the method of denuding.

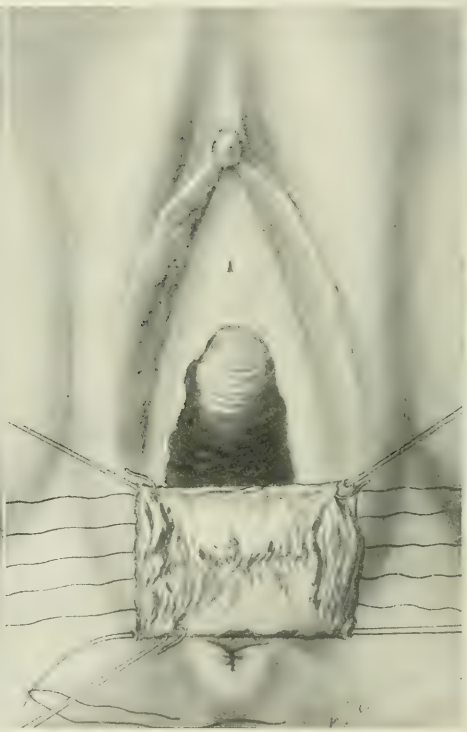

FIG. 29.—Tait's operation for complete laceration through the sphincter; the method of passing the sutures.

rectovaginal wall as far as the base of the denudation, is, in reality, the forerunner of all these methods of direct sphincter-suturation, and, I believe, to Boulton belongs the credit of this very valuable improvement in the technic of the complete operation.

One of the most valuable of the recent modifications was suggested by Hirst.[40] He introduces four septal sutures of catgut from the rectal side, knotting them in the rectum. "Above the rectal sutures a triangular stitch of silkworm-gut is inserted somewhat resembling the Emmet suture for securing the ends of the sphincter, but placed above the sphincter, which is brought together by the rectal stitches; this suture acts as a splint to the rectal wound, an additional support to the sphincter stitches, and a barrier between the rectal and vaginal sutures. The vaginal wound is united by vaginal and perineal sutures of silkworm-gut as in the Hegar operation for lacerated perineum."

G. H. Noble [41] has devised a very ingenious operation which converts a complete into an incomplete laceration. His method consists in splitting the rectovaginal septum, dissecting the lower end of the rectum from the vagina, and drawing its anterior wall down through and external to the anus. The protruding rectal flap is anchored in the perineal incision. This avoids the introduction of rectal stitches.

THE FLAP-SPLITTING OPERATION IN COMPLETE TEARS OF THE FLOOR

The Tait operation for complete tears of the perineum differs but slightly from the operation as already described for incomplete lacerations (Figs. 28 and 29). The rectovaginal septum is split as before. The dissection is carried to the vertical incisions which are now extended posteriorly so as to form an H-shaped incision, the shorter portion of the vertical cuts lying posteriorally to the transverse bar. The upward and downward dissections are made as extensive as the case may require. The two flaps when drawn apart form a quadrilateral opening, at the depth of which lies the undivided portion of the septum. The first suture, as in the Emmet operation, must be made to traverse the sphincter ends. All the sutures are introduced from the perineal surface.

[40] Univ. Med. Mag., Jan., 1899.

[41] Jour. Am. Med. Asso., Aug. 9, 1902.

GONORRHEA IN WOMEN

A POST-GRADUATE LECTURE DELIVERED AT THE SAMARITAN HOSPITAL FOR WOMEN, LONDON

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GENTLEMEN: Within our recollection a few diseases have been described and brought into prominence that were unheard of when we entered upon the study of medicine, and such diseases are regarded—by the lay mind at all events—as "new." Whether there is such a thing as a new disease, or not, I will not venture to discuss at the present time, but one thing is certain,—the disease about which I have to speak to-day is probably as old as history itself; at any rate we know that it dates back as far as the ancient Israelites and Egyptians. In spite of its antiquity and, I may add, its ubiquity, gonorrhea is scarcely mentioned in the older text-books on gynecology, and, what is perhaps even more striking, no modern treatise on diseases of women contains a chapter devoted entirely to this important subject.

Whilst gonorrhea has generally been regarded as an etiologic factor of vaginal catarrh, until the year 1878 gynecologists had no means of distinguishing between simple and infective catarrh in women. A certain amount of secretion is normal, and catarrh is very frequent; but before Neisser discovered the specific cause, it was impossible to understand why in one case the discharge could be transmitted, whilst in another it could not. As late as 1872 we find that there were two schools,—the one that denied the specificity of gonorrhea, and thought that every mucous membrane could take on suppurative inflammation and then afford an infective secretion, and the second school, that believed in a specific poison but could not prove it, the diagnosis being influenced by such external conditions as the respectability or non-respectability of the patient—whence gonorrhea was often diagnosed when it was absent and not diagnosed when it was present.

Neisser has shown us that the specific character of gonorrhea is due to the presence of a diplococcus which is distinguished by its shape, by its behavior to various staining reagents, and its power of penetrating white blood corpuscles. It consists of two cocci divided by a narrow clear space toward which they are slightly bent, thus giving them the shape of two closely-apposed kidneys. Compared with other cocci they are relatively large in size. They multiply by rectangular division, producing clusters but never chains. The fact that it is decolorized by Gram's iodine solution distinguishes the gonococcus from all other common cocci. Its presence inside a pus cell is pathognomic.

The diplococcus of Neisser has specific pathologic peculiarities that distinguish it from all known organisms. How were these properties acquired? Bumm says, "Perhaps for once, in a way, an originally non-pathogenic organism, under given influences, bred the peculiar potentialities of the gonococcus," that is, he seems to apply the theory of evolution to parasitic life.

Of the peculiarities above referred to, the fact that the gonococcus shows a selective parasitic adaptability for the human race is the most striking. It is only to be found on, or in, or beneath human mucous membranes; even the highest apes cannot be infected. I have tried all over London to obtain a pure artificial culture in order to show it to you this afternoon, but without success. This is no doubt due to the fact that this germ is most difficult to cultivate outside the human body. On human serum it will develop an active growth through many generations. It will also grow on peptonised nutrient media, in cystoma fluid, ascitic fluid, and sparingly in urine agar. These growths, though very resistant to heat and cold, soon die. The diplococcus is killed by drying, so that inspissated secretions are not infectious. Moist germs live for many hours in cold boiled water and for five minutes in antiseptic solutions of the ordinary strength used in surgical practice.

The gonococcus has a more uniform virulence than most pathogenic germs. Wertheim took cultures from a chronic case and by injection produced intense gonorrhea; cultures from this experimental case, when injected into the chronic case, used in the first instance, produced acute symptoms.

The danger of gonorrhea is very largely due to the power pos-

sessed by gonococci of penetrating uninjured epithelial tissues. Once underneath the surface, the tissues afford them nutrition and protection, whence the chronicity of the disease and its frequent latent characters are fully accounted for.

There is no congenital immunity against gonorrhea nor can such be procured. The wife to whom a husband has given the disease may re-infect her husband. Chronic cases are as dangerous as they are common, owing to the fact that gonorrhea may be present in chronic form without subjective signs to warn the infected person of its presence.

Gonorrhea can be very obstinate, but it can be cured. Many bacteriologic investigations are necessary, however, before we are in a position to pronounce upon a cure. The average incubation period by the natural mode of conveyance of the organism is about three days. Accurate determination of the site of infection in women is a matter of prime importance for the gynecologist. This will depend, to some extent, upon the anatomic peculiarities of the genitalia. A highly situated urethra is more likely to escape initial infection than one which opens lower down; a low cervix with a gaping vagina may lead to the former being the first site of inoculation, but a very common occurrence is to get both urethra and cervix infected at the same time.

Gonorrhea of the Urethra.—This is extremely frequent, as it is generally present in fresh infections. The surgeons at the Lock Hospital see more of it than we do. In three days the discharge is purulent, the swollen lips of the meatus present as prominent red flaps, owing to the ectropion of the mucous membrane, and between these, a drop of greenish-yellow pus exudes. The canal is tender up to the neck of the bladder; on endoscopic examination the swollen mucous membrane is seen to be covered by pus and a croupous exudate, and on removal of the latter a slightly hemorrhagic red surface is disclosed. The patient's general condition is not much interfered with; subjectively there is tickling or burning after micturition.

Then follows a dull, continuous, pressure sensation above the symphysis, or cramp-like pain during micturition may be complained of. These subjective symptoms soon lessen; in three or four weeks the urethra has its normal appearance, and in from

six to twenty weeks it heals. If the general state of the patient is good it may heal spontaneously; if anemia, chlorosis, or struma be present, it may pass into a subacute or chronic stage in spite of treatment. In the chronic stage gonorrheal urethritis is difficult to detect; if the patient has voided urine within an hour of examination, pus may be absent. In this way a woman who may be regarded as clean by her doctor may infect her husband. An important point to know is that even when pus is absent from the urethra the swollen meatal lips present for some time as red or gray jelly-like elevations, very sensitive to the touch, and they bleed on palpation. Later on, the whole tube feels thick and hard, and internally the mucosa looks granular or jelly-like, plaques of matted gray or glassy appearance may be seen. Scar contraction and urethral stenosis, as well as muco-purulent catarrh of the bladder, may result. Chronic gonorrheal urethritis may at any time become subacute or even acute.

In the lower part of the urethra and near its mouth are found tiny glands, the homologues of the prostate; these are the glands of Skene or the para-urethral glands. Their lacunæ soon become infected in the acute stage of gonorrhea, their ducts get blocked, and small miliary abscesses form at the sides of the meatus. It is important to remember these glands in the treatment of gonorrhea in women.

Gonorrheal Inflammation of Bartholin's Glands.—This is often double in acute stages of the disease, and generally so in the chronic form. The rule is for one gland to become infected before the other; it does not always result in abscess formation and before it does so the appearances are not very striking. It is detected by the extroverted swollen mouth of the duct from which pus can be squeezed; and a spindle-shaped hardness indicates adenitis of the gland. It is as well to give here a word of caution about an over-readiness with some people to diagnose gonorrhea from undue redness of the orifice of Bartholin's duct. This feature may be due to innocent causes, and, although it was designated the "macula gonorrhœica" by Sänger, it is by no means pathognomonic of this disease. Sometimes the duct becomes blocked with a cast swarming with cocci. Specific Bartholinitis may last for many months, giving exacerbations especially after coitus. It gives a

dull pain on walking or sitting, but subjective signs are not prominent. In chronic cases the duct may be blocked partially or completely, the secretion becomes pent up in either case, but if the block is only partial the secretion escapes, and fills up again, giving what Martineau called "abscess à répétition." If the block is complete and the secretion great, a large abscess, the size of a pigeon's egg, forms. This is situated at the hinder part of the labium majus and pushes forward as far as the vestibule; it is fluctuant, and the skin over it is red, stretched, and shiny. It ruptures in from one to two weeks, generally on the inner side, seldom above or below; a suppurating fistula is formed which gradually closes up. In the pus no gonococci may be found nor bacilli of any kind. It should be specially noted that not every cyst nor even abscess of Bartholin's duct is necessarily of gonorrheal origin. Pyogenic staphylococci may enter under certain conditions, especially if the ducts are unusually patent from chronic inflammation; then an abscess results, which is much more tender and painful than that due to the activity of the gonococcus, and in the pus the staphylococcus pyogenes aureus will be found.

Gonorrhea of the Vagina.—The vagina was formerly regarded as the commonest seat of this disease, but this is only true of children; in adults it is not so, and for a reason not far to seek, if we bear in mind the thickness of the vaginal epithelium. This thick keratoid vaginal lining offers considerable resistance to the entry of penetrating diplococci, whereas the later find it a comparatively easy task to enter through the single layer of cubical epithelium found on the mucous membranes of the urethra and cervix uteri. In children, the vaginal epithelium is thin and soft, and hence more easily penetrated by the cocci. With regard to the share which the adult vagina takes in the process of gonorrhea infection, I feel I cannot lay too great stress upon the fact that in chronic gonorrhea there is nothing in the appearance of the vagina at all suggestive of its presence, simply because the disease is not in the vagina, but in the cervix uteri and urethra; we must lay no stress upon the appearances of the vaginal walls in the diagnosis of chronic gonorrhea. In young people and nullipara, redness and swelling of the vaginal mucous membrane are met with often enough in the early stages of infection. This is generally due to second-

ary invasion by pus from the cervix. In children, gonorrheal vaginitis is the usual result of infection.

True primary gonorrheal vaginitis in adults is a most exceptional occurrence. It presents very striking features. There is such an abundance of pus that the outer genitalia are matted by dried secretion; the small labia are red, edematous, and project beyond the labia majora, and they are eroded on the vaginal surface. The hymen is likewise swollen, tender, and eroded; patches of adherent lymph are seen here and there, whilst every furrow is filled with pus; the whole mucous membrane easily bleeds on touch, and is so exquisitely tender that examination has to be conducted under a general anesthetic. The symptoms are marked—pain, heat, burning, and stretching of the outer parts, tenderness of the whole lower abdomen, inability to walk, even sneezing, coughing, and loud speaking being painful. There is fever with a few rigors. The symptoms subside in about three weeks, if no menstrual period intervene; it may leave behind a chronic cervicitis, but it never leaves a chronic gonorrheal vaginitis. The patchy "vaginitis granulosa," which runs such a chronic course is not due to gonorrhea; it gives an abundant secretion but no gonococci will be found in it. Martineau regards it as a scrofulous or rheumatic condition.

Gonorrhea of the Uterus.—Next to the urethra the cervix is the most common seat of infection, which may be direct, or through the folds of mucous membrane which runs from the posterior lip of the cervix to the vaginal wall. The cervix is swollen, the mucous membrane is stretched, shiny, and red, and through it present suppurating points of infected Nabothian follicles, giving the vaginal surface of the cervix an acneiform appearance. Bumm regards this last condition as a sign of repeated infection. Pain is not a well-marked symptom, movements of the uterus cause a slight tenderness, the patient complains more of the discharge and scalding than anything else, but in some instances there is pain in the back and over the pubes.

Gonorrhea of the Cavity of the Uterus.—This gives symptoms of acute metritis. In the early stages the cervix cannot be drawn down without pain. Under an anesthetic the body is found to be only slightly movable on the cervix; it is stiffly anteflexed or retroflexed, there is a sense of fullness and stretching,

with a boring or pulsating pain in the pelvis, leading to a dreadful cramp-like "bearing down." Bodily movements are impossible, and the patient feels very ill. Fever is already present.

Young women whose first attack reaches the cavity of the uterus always suffer most, that is, more than those who have suffered from preëxisting chronic cervicitis. It is a notable fact that the severe symptoms rapidly subside. In a week, or sometimes a couple of days, the tenderness is less and the cramps are gone, the patient gets about again, and attaches but little importance to her few days illness. We do not, as a rule, meet with acute uterine gonorrhea in gynecologic practice. These cases come to us after the acute attack is over, when there is nothing pathognomonic in the symptoms; the secretion is often so slight as to be easily overlooked; this applies especially to chronic infection limited to the cervical canal, the only symptom of which is discharge, which may be quite clear whilst still containing cocci. Erosion of the cervix may be present, but more suggestive than erosion is a bright red inflammatory border to the orifice, with some ectropion, and with a history of exacerbations, especially after sexual excess or menstruation, under which circumstances the flow again becomes purulent and the pelvic pain returns.

Chronic gonorrhea of the cavity of the uterus which has not trespassed to the tube has no special symptom of its own, and can only be diagnosed by the presence of gonococci in the discharge; but in some, especially the more chronic cases, there arise signs of chronic metritis: the uterus is large and tender, the mucous membrane is hypertrophied, the menses become too frequent and too profuse, the patient develops nervous symptoms, she is now better and now worse. When once chronic, gonorrheic metritis may last for years, and it is almost impossible to assure a patient that she is ever cured. Nevertheless spontaneous healing does occur.

Gonorrhea of the Tubes.—Fortunately for the sufferers gonorrheal infection generally stops at the internal os uteri, but, as we have just seen, it can reach the cavity of the uterus, and in like manner it can trespass beyond the natural barriers afforded by the narrow opening of the tube and so give rise to gonorrheal salpingitis. This is especially prone to happen in the late puerperium.

It is ushered in by acute symptoms and is sudden in onset. It can start months or even years after the initial infection. Should the uterine inflammation already have subsided, the signs of tubal infection are all the more striking and localized. The onset of infective salpingitis is not infrequently accompanied by a fresh flow of pus from the uterus, but this is not invariable. There is an initial rigor, with fever, which within a fortnight drops to normal; there is deep pelvic pain of a continuous boring character; as the condition is often bilateral, there is tenderness in both groins over the eleventh dorsal segment; pain is elicited on pressure and by any movement of the body, and even shaking of the bed calls it forth. Menstruation is rendered painful, crampy, more frequent, more excessive, and it lasts longer. In acute salpingitis the abdominal walls are too rigid to make out more than a tenderness on one or both sides, but with the aid of an anesthetic the tubes feel like stiff strings running outward and backward from the uterine cornua; an increase in their size is not at first noticeable. Healing can now set in, and the possibility of a future pregnancy is not yet excluded. If complete healing fails, the tenderness on pressure persists, menstruation continues to be painful, the woman is sterile, and liable to periods of relapse. The uterus may be found in a state of fixed retroflexion, there is a mucous discharge, in which gonococci are absent. With the climateric, the pain subsides The results of chronic gonorrheal salpingitis are therefore sterility and pelvic pain. But this is not all, for a large pus sac may develop with involvement of peritoneum, ovaries, and surrounding viscera; this is indicated by a corresponding increase in severity of the general and the local symptoms. It is not usual for a tubal abscess to arise directly on infection, there is generally a history of frequent attacks with improvement in between, but the final effect is the clinical picture of a pale, wasted, prematurely-old woman, with sunken eyes and sickly aspect, who, in addition to various nervous disturbances, complains of sacral and pelvic pain, anorexia, dyschaesia, and a chronic white discharge—the picture, in short, of a woman who lost all pleasure in life. Her menstrual periods are irregular, they come too soon, and often at these times a fresh disturbance, in the shape of a spread of peritonitis, is lighted up. Examination may start a fresh attack of fever and pain; otherwise

there will be no fever. One finds in these cases a tumor of variable size lying in the pouch of Douglas, only slightly mobile, often quite fixed, and tender on pressure; this tumor feels larger than it really is, owing to adherent omentum, adnexa, and small bowel, and also to the fact that it may be encapsuled in the densest adhesions.

When once a pus sac has become thoroughly encapsulated in dense adhesions the acute attacks of pain and fever are less frequent, and if the patient will not submit to operation she may drag out a weary life of ill health for many years.

Gonorrhea of the Ovaries and Peritoneum.—The escape of pus from the tube to the surface of the ovary leads to the development of gonococci in the germinal epithelium; thence they invade the follicles and cause pus cysts in the ovary. This cannot be independently diagnosed, because the symptoms of gonorrheal ovaritis are masked by those of salpingitis. Wertheim states that infection of the ovary can occur by deportation of the germs along the lymphatics as well as by their transmission along the surface by the injection of pus through the ostium of the tube as mentioned above; but it may be taken for granted that ascending gonorrheal infection is usually a catarrhal process, the cocci being distributed by direct continuity of surface tissues. Bumm has proved this by showing that the gonococci are rarely found deeper than the submucosa, and that they readily die when they reach the true connective tissues; nevertheless such terrible conditions as gonorrheal endocarditis and gonorrheal pyemia, in spite of the fact that they are generally mixed infections, strongly favor Wertheim's view that gonococci can be deported by means of the lymphatic stream.

In acute ascending gonorrhea, even when there is no escape of cocci from the abdominal ostium of the tube, there occurs fibrinous exudation of the tubal and uterine peritoneal coats. The thin wall of the tube allows the spread of inflammation by continuity of tissue to its own serosa. This kind of inflammation may entirely subside. Of more importance are those cases of peritonitis due to the escape of pus from the tube. Now, gonococci do not live very long on the peritoneum, but they exist long enough to cause a fibropurulent exudation on the surface, the final results of which are mattings of the neighboring organs, tubes, ovaries, omentum, rec-

tum, and parietal peritoneum. Since the gonococci quickly die on the surface of the peritoneum, each attack of gonorrheal peritonitis is localized; it spreads only by repeated fresh attacks, and general peritonitis, such as we see in septic cases, never occurs. The fibro-purulent exudation, with subsequent formation of adhesions, is nature's mode of defence, since it shuts up the tube and encapsulates the pus. Straining at stool, bending of the body, and over-reaching, may rupture adhesions which have formed, and allow the escape of pus to a healthy peritoneal surface.

The onset of peritonitis is clinically indicated by increased pain, and some patients are extremely ill as soon as the peritoneum is involved. The severe pain at the onset and all future attacks are explained by peritonitis.

Gonorrhea of the Anus and Rectum.—These are rare occurrences. We find gonorrheal pus around the anus without the latter being infected. Direct contact of pus with the deeper parts of the anal canal is necessary to cause the disease. It is caused by coitus per anum, unclean instruments, and by the finger. Rectal gonorrhea is more frequent than is supposed; a perineal tear leads to this condition. The symptoms are heat and burning after stool, with the formation of erosions, ulcers, and fistulæ at the anal orifice. The secretion may be mixed with blood, edematous folds of mucous membrane hang dependant at the anal ring; these are very tender to touch. When the disease has become chronic, the swelling subsides, but the mucosa is still red, and on dilatation, erosions can be found above the anal orifice and even above the internal sphincter. It is difficult to get the cocci out of the mucous folds and hence rectal gonorrhea is prone to become chronic and is capable of leading to granulation and stricture. In women the latter is usually situated below the internal sphincter when due to gonorrhea. Periproctal abscess and rectal fistula may result from rectal gonorrhea.

The Course of Gonorrhea in Women.—The disease may be slight or severe. As the gonococcus has a standard virulence the difference lies in the localization of the infective process. It is slight so long as it is confined to the urethra and cervix; once beyond the internal os, the disease becomes severe. Many women date the onset of their illness from this moment. The condition which is often overlooked is chronic urethritis, with or without

cervicitis, the acute stage is soon over and the pain forgotten, a slight discharge is left, which is ignored, the women is acclimatised, and does not consult a doctor. Chronic urethritis and cervicitis may spread without coitus.

There are two mechanical barriers to the ascent of gonorrhea: the internal os and the uterine end of the Fallopian tubes. The cocci have no power of movement in themselves, their spread is by passive deportation; the physiologic stenosis of the os and the minute calibre of the interstitial part of the tube are not sufficient to obstruct so small a body as a coccus, but yet small enough to obstruct mechanical deportation. Of functional causes which overcome this obstruction and which favor the spread of the disease, menstruation is of primary importance; hence we not infrequently see the severe symptoms characteristic of gonorrheal endometritis set in at the end of menstruation. The puerperium works in the same way, so that in the first week the germs may rapidly spread. Excessive and prolonged exercise during menstruation, especially such as stooping and lifting heavy weights, and also jumping, are all dangerous. Another cause for spread is excessive venery, more particularly just before or after menstruation; lastly comes therapeutic interference involving the introduction of instruments into the uterine cavity. Many inflammatory conditions following the use of the sound are not of a septic but gonorrheal nature. In the spread of the tube from the uterine cavity, the menses, puerperium, excessive exercise, etc., come into play as before; the process is not always simultaneously bilateral, so that one tube is found in a state of purulent catarrh whilst the other is a pus sac.

Functional Disturbances.—These vary with the localization. Gonorrheal disease of childhood does not disturb later functions because the genital apparatus is not yet opened up, and so the uterus and tubes escape; any after history relates to vulval conditions, the symptoms being itching and burning. The course of gonorrhea in an otherwise healthy child is favorable, the disease is curable in from four to six weeks. Functional disturbances in adults relate to menstruation and ovulation. Gonorrheal endometritis and salpingitis disturb menstruation, the interval is reduced, the duration of the flow is prolonged, and the amount is increased, dysmenorrhea of all kinds is produced; it may commence

a week before the flow and last as long afterward. Salpingitis and peritonitis cause exacerbations of menstrual pain. These irregularities vary in different individuals, and in one and the same person at different times. Ovulation is not interfered with until the ovary becomes encapsulated by adhesions and suppurates, or is in a state of fibroid induration throughout. In the latter case the periods are postponed and there is a tendency to amenorrhea.

Relation of Gonorrhea to Fertility.—Gonorrhea by no means always leads to sterility. It does not do so in the majority of cases. The frequency of ophthalmia proves this, since the disease was present in the mother at the time of conception in most cases. Conception can even occur after salpingitis. Still, many women are rendered sterile, either completely, or after the birth of one child. We can easily understand this in the case of pus tubes, or after closure of the tubal orifices, but in some of the cases of sterility the tubes and ovaries appear normal; these are not so easy to explain. The relation of gonorrhea to abortion is not definitely settled. Sänger held that gonorrhea of the endometrium produced abortion about the fourth or fifth month from inflammation of the decidua, and gonococci have been found in the decidual fragments, which formed part of an abortion. After the ovum has completely filled the uterine cavity, gonorrhea will not produce abortion, there will be no symptoms as far as the uterus is concerned, but there will be a very profuse discharge both from the cervix and from the urethra.

Gonorrhea in the Puerperium.—In pure gonorrheal infection where other germs are excluded, the cocci are very abundant in the lochia, the puerperal process favors their growth; the fever in the early puerperium is not, as a rule, high unless the infection is mixed. It is in the late puerperium that the tendency to ascending gonorrhea arise. When fever and local pelvic peritonitis develop late in the puerperium, think of gonorrhea.

Mixed Infection and Metastases.—Staphylococci and streptococci may gain access during the course of gonorrhea. When this occurs the character of the disease is completely changed. Suppuration of connective tissues can set in, with the development of parametritis and pyemia. The above organisms in association with Bacillus coli have been found in the vegetations taken from

the cardiac valves in cases even of malignant endocarditis. Gonorrheal arthritis is less frequent in women than in men. One woman to sixty-four men is the average given by Bumm. In women the affection is generally monarticular and afebrile; the knee is the joint of election, the lesion is a hydrarthrosis. Women may develop polyarticular rheumatism and hyperpyrexia.

Statistics and Prognosis.—As regards the frequency of gonorrhea, Noeggerath says that of a thousand married men in New York, eight hundred have gonorrhea, and in 99 per cent. of all these the gonorrhea remained uncured; therefore in New York three out of every five married women have gonorrhea. As regards the question, Is gonorrheal ever cured? Bumm collected 53 cases with 36 cures; he thinks most cases are cured. Schwarz says that in large towns 10 per cent. of men marry whilst suffering from chronic gonorrhea, another 10 per cent. acquire a fresh attack during married life.

One woman in every five contracts gonorrhea as the result of marriage. In the obstetric wards at Heidelberg, Opphenheimer found gonorrhea in 30 out of 108 cases of pregnant women, that is 27 per cent. Schwarz's percentage was 12.4 in pregnant women, and this agrees with the proportion of ophthalmia met with in new-born children. Noeggerath found that, in 81 cases where the husbands had gonorrhea, 49 wives were absolutely sterile and eleven more were relatively so, that is, they never conceived during a period of three years from the date of their first pregnancy. Glünder found that in 87 sterile women 71 had gonorrhea. "Eight per cent. of all women are sterile from gonorrhea," says Bumm.

Diagnosis.—This is much more difficult in women than in men. Discharge is a symptom of a whole series of pathologic conditions which are not infective. If on vaginal examination we see pus on squeezing the urethra, and if at the same time there is redness and ectropion of the urethral mucous membrane, it is safe to diagnose gonorrhea. In the chronic stages there may be no pus in the urethra until some hours after micturition. Pus or muco-pus from Bartholin's gland proves gonorrhea, but the redness of the orifice of the duct, which Sänger called the "macula gonorrhœica" is of no diagnostic value. Suppurative adenitis generally means gonorrhea, but not always, since puerperal women may get it from sepsis.

Gonorrhea of the vulva, vagina, cervix, body of uterus, and tubes, cannot be diagnosed with certainty by clinical means.

Pus may be harmless, mucus may be virulent. In adults a purulent discharge coming from the vaginal walls or from the vulva is generally not gonorrheal. In the same way the characters of the mucous surface show nothing. Granular vaginitis is not generally gonorrheal; the same applies to erosions and various inflammations on the cervical mucosa. Even the "gonorrheal warts" are not always pathognomonic. All the changes which can arise in the uterus, tubes, and peritoneum from gonorrheal infection can also arise from sepsis, especially in the puerperal state, therefore, we must take full account of all associated conditions, and gauge the history carefully, in order to diagnose gonorrhea with safety. In cases of young women who are unmarried, tubercle must be considered in the differential diagnosis of tubal swellings. Threads in the urine are suggestive but not pathognomonic.

Scalding micturition with a vaginal discharge arising soon after marriage is probably due to gonorrhea.

In all cases of suspected gonorrhea the discharge should be collected on a sterile swab from the cervix or urethra, or from the duct of Bartholin's gland, and should be transferred to a clean cover glass. It is then reduced to a thin even film by drawing another clean cover slip across it, and then both smear preparations are allowed to dry in air. Next pass them through a spirit flame. Stain one side with Loeffler's methylene blue, and the other with Gram's preparation. Dry and mount in Canada balsam. The cocci are recognized by their characteristic size and grouping, and by taking the blue stain deeply, but what is most important they are decolorized by Gram's iodine solution. If we find diplococci in pus cells which refuse to retain Gram's stain we are in a position to make an absolute diagnosis of gonorrhea.

Prophylaxis.—The public must be taught to regard gonorrhea as a serious disease and not as a trifling accident or a slight misfortune. Doctors must warn their male patients of the serious results of this disease in married life. Marriage during the existance of chronic gonorrhea should be forbidden. Prostitution should be controlled, prostitutes should be retained in the hos-

pital until all gonococci disappear. Only in this way can the principal source of gonorrhea be stopped. Patients should be warned of the infectiveness of the discharge, sponges should never be used for its removal, cleansing of the genitals must be done with wool, which can immediately be burned.

Treatment.—In the acute stage the patient should rest in bed, take a sitz bath, and wash the external genitals with 1 to 1000 solution of mercuric bichloride. Employ no local treatment in the acute stage; do not pass fingers, speculum, Playfair's probe, or sound. Smear the external genitals with lanolin to prevent excoriation by discharge. In cervical gonorrhea, in the early stages, do not paint the cervix, but keep the vagina clean with douches and baths. After the fourth week, when the condition will have become subacute, paint the endocervix with a Playfair's probe dipped in 5 per cent. ichthyol, or 5 per cent. protargol, or 2 per cent. argonin.

For urethritis, in subacute cases, frequent daily applications of 1 per cent. protargol, carefully injected by means of a small syringe under low pressure, should be used. Copaiba balsam does not kill gonococci, but empirically it does good, especially in gonorrhea of the neck of the bladder. It may act on the cocci indirectly through the tissues. True gonorrheal vaginitis, which is rare in adults, should be treated by a douche of 1 per cent. ichthyol and baths.

In chronic gonorrheal urethritis use the endoscope and paint any erosion with 20 per cent. silver nitrate, or apply the galvanocautery. For infected follicles about the vulva and meatus urinarius, use the galvanocautery, introduce the point cold and gradually turn the light on. Bartholin's duct should be split and cauterized with stick silver nitrate. Extirpation of the gland and duct, if a retention cyst or an abscess develops, must be carried out.

For chronic gonorrheal cervicitis, draw down the cervix with the volsellum, paint the canal with 5 per cent. solution of argentamin or silver nitrate until the surface becomes filmy white, and leave an ichthyol tampon (10 per cent. in glycerine) in the vagina for twelve hours. Treat gonorrheal erosions of the os with lunar-caustic.

Gonorrheal endometritis must be treated with great caution. Do not curette, except for severe menorrhagia, which is very rare. Do not drag down the uterus forcibly with a volsellum.

Apply a 1 per cent. solution of protargol or silver nitrate on a Playfair's probe; leave it in utero a few minutes, then continue the treatment by douching the uterine cavity with 0.5 per cent. silver nitrate, introduced by means of a two-way catheter, the patient of course being kept in bed. If these weak solutions fail, in obstinate cases paint the endometrium with strong iodine solution. In any case if fever follows from intrauterine treatment, the treatment should certainly be discontinued.

For salpingitis, absolute rest in bed, cold compresses to the abdomen, and morphin suppositories per rectum, with saline purgation must be employed. Prolonged rest is the most important matter to attend to, it should be continued for two months, at least; in the case of a recent attack, the patient should not be allowed to walk until the temperature has been normal for one month.

If a pus sac develops, it must be removed, perferably through the vagina, and the uterus should be taken away at the same time, for, if it is left, the cornual stumps are a source of infection.

For gonorrhea in pregnancy, coitus should not be indulged in; the treatment should be confined to douching. If abortion occurs there is great risk of ascending gonorrhea. Do not irrigate, but be content with baths and washing. In parturition, complicated with gonorrhea, do not make vaginal examinations.

During the puerperium, prolonged rest is most essential. Keep your patient in bed four or five weeks, even if there has been no fever, always remembering that the time of greatest danger for the puerperal woman, who is unfortunate enough to be the subject of gonorrhea, is when she first attempts to get out of bed and move about. By insisting on our patients spending an extra fortnight or three weeks in bed in these cases we shall save many from much misery and by this simple means rescue some from serious danger to life.

TWO CASES OF ECTOPIC PREGNANCY WITH SOME UNUSUAL FEATURES

A CLINICAL LECTURE DELIVERED AT THE KENTUCKY UNIVERSITY MEDICAL DEPARTMENT

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GENTLEMEN: I am fortunate this morning in being able to bring before you two women, both negresses, suffering from conditions alike in character, that is ectopic gestation, but presenting features of unusual interest. While such cases are not infrequent, they always possess much interest, not only because of the abnormality itself, but particularly from a pathologic standpoint when the condition has existed as long as in the patients to be presented. The diagnosis is oftentimes exceedingly difficult, as will be amply illustrated by the first case shown. The etiology also merits serious attention and furnishes a fruitful source of speculation.

CASE I.—I am permitted to show the first patient through the kindness of Prof. J. M. Holloway. The patient states that she is 28 years of age, married to her second husband, and that she has had two children, one being now 10, the other 5 years old. Between the birth of these two children there were no pregnancies, nor has there been any evidence, so far as we can ascertain, of conception following the birth of the second child until a few months ago, when she began to complain of the condition for which she now seeks relief.

You will observe that five years elapsed between the first and the second pregnancies, and five years between the birth of the second child and the development of the present symptoms. Her second marriage took place twelve months ago, and she menstruated once a short time thereafter. Menstruation not recurring at the regular time, she believed herself to be pregnant. Being a young, healthy negress, of course, such was the natural assumption. She had

morning sickness, the abdomen gradually increased in size, and preparations were made for the birth of a child, which event should have occurred two and a half months ago. Upon inquiry we elicit the fact that during the fifth month of pregnancy she suffered acute pain in the left side, which caused her to remain in bed a day or two. She says the pain was not especially severe, but fearing a miscarriage, and wishing to present her second husband with a child, she was unusually careful—for which reasons, rather than on account of the severity of the pain, she remained in bed.

At what she presumed to be the end of her pregnancy, and what would have been the termination of normal utero-gestation, she began to have labor pains accompanied by a bloody discharge from the uterus. A physician was hurriedly summoned and preparations made for prompt delivery. After an examination the physician told her she was in labor. However, the child not being born within a few hours, a second examination was made, and she was then informed that there was an obstruction which prevented delivery of the child, that he (the doctor) could do nothing more for her and he left. The pains continued for some time as did also the sanguineous uterine discharge, but at the end of two weeks both had ceased. She felt active fetal movements during this period, but all motion ceased when the discharge disappeared. The abdomen then began to decrease in size, the diminution being quite noticeable within the next three or four weeks. There remained, however, considerable abdominal enlargement which you will observe is still present, and this is why she sought relief. A physician at her home, so we understand, made an x-ray examination, but could discover nothing abnormal. She was then brought here and has been under our observation for several days.

The history is indicative of ectopic gestation with missed labor; but is not altogether typical, in that there has been entire absence of pain such as would be expected if rupture of the tube had occurred into the general abdominal cavity or the folds of the broad ligament,—a requisite for further development of the fetus, since the tube cannot enlarge sufficiently to accommodate a full-term child. Moreover, the atypical contour of the abdomen and the fact that we are unable to detect any of the limbs of the fetus (assuming that one is present), although it has evidently been dead but a short

time, contribute to the difficulties encountered in arriving at an accurate diagnosis.

If not an ectopic gestation, then what could be the condition present in this case? The woman maintains that she is pregnant, or at least has been pregnant, and that having had two children she knows what the symptoms of pregnancy are. We must give some credence to her statements. I believe this tumor to be the result of an abnormal pregnancy, but I am not positive. What else could it be? Might it be an ovarian tumor, possibly dermoid in character? The clinical history is not indicative of ovarian cyst. There is no fluctuation, nor is there any evidence of an inflammatory condition such as might result from infection of a dermoid, causing it to increase to these proportions. I think we can exclude ovarian cystoma. Might it be a fibroma? This seems more probable, since the uterus has largely increased in size and, by bimanual examination under chloroform, cannot be separated from the tumor; in fact, the mass appears as a part of the uterus, moving with it and *vice versa;* the growth is irregular in outline, as can be clearly demonstrated with the abdomen perfectly relaxed from effects of the anesthetic; it is somewhat nodular, and is crowded into the pelvis, being more or less fixed, though susceptible of some motion. Again, it must be remembered that this woman is a negress, a race peculiarly prone to fibroid tumors, my experience (which in this clinic has not been small) demonstrating that they occur three or four times as frequently in the black as in the white race. But how can we account for the absence of menstruation, the fetal movements, the morning sickness? These phenomena may all be brought about in certain cases, as you have seen illustrated, through the influence of the nervous system. Some of you will recall that I showed you not long ago a case of pseudocyesis, in which there were present abdominal enlargement and other symptoms such as would result from a true pregnancy.

One point in the history of this case that I have neglected to mention is that the woman still has some colostrum in her breasts. She has had decided mammary enlargement, but this, and even the secretion of milk, may accompany tumors involving the generative tract. So the question is not entirely easy of solution, and while, as already stated, I believe the patient has an ectopic gestation

which has gone to term, that there has been a spurious (or missed) labor, that the child is dead and now exists as a foreign body in the abdominal cavity, I am not absolutely certain. However, we shall shortly ascertain and be able to demonstrate just what is the condition present. One thing we know is that the treatment which should be pursued is clearly indicated, and our operation will in that sense be more or less exploratory.

Incising the abdomen we come down at once upon the large tumor, above which is the transverse colon, and over which course the large blood-vessels going to the colon. The descending colon lies in part over this tumor. The right tube and ovary are adherent to the tumor. The bladder is observed in front, and rising behind it the fundus of the uterus can be plainly seen. There is an enormous tumor-mass which seems to be covered with peritoneum. The tumor will be quickly incised, taking care not to interfere with the large blood-vessels. Our prediction that the condition was one of ectopic gestation is now confirmed, as can be readily noted. We will simply sever the cord and remove the fetus, and as the placental vessels are undoubtedly obliterated there will be little danger of hemorrhage from that source. The entire gestation sac will then be removed.

This tumor lies behind the peritoneum; in fact the child has developed retroperitoneally; I must therefore exercise considerable caution in our enucleation that I may not injure the ureters, that I may not interfere with the blood supply to the colon, and that I may not seriously damage the intestine. Everything above having been separated I proceed to remove the placenta, a portion of which can be seen lying to the right side and adherent to the uterus and the broad ligament; but I am sure I shall not be able to accomplish this without performing complete hysterectomy. I shall therefore begin on the right side and remove the uterus and this mass *in toto*. I first ligate the ovarian vessels at the infundibulo-pelvic ligaments, separate the broad ligament, ligate the round ligament, and cut across the uterus—grasping and ligating the uterine artery as it spurts. No difficulty is experienced in peeling out the mass, and the hemorrhage you will note is trifling. It is impossible, however, to establish a pedicle for the right ovarian artery, as I am unable to isolate the infundibulo-pelvic ligament on that side, so I merely

transfix the mass, pass a ligature around it, taking care not to include the bowel, and cut it away (Figs. 1 and 2).

The mass I have removed includes the fundus and body of the uterus, the right ovary and tube, the left ovary and tube, and the gestation sac. The peritoneum is brought together with catgut, and I place a little gauze over a small area of the bowel which is slightly damaged and which I have carefully repaired with catgut, and then close the abdomen, placing a drainage tube with a gauze canal at the lower angle.

Examination of the specimen shows that the ampulla of the tube is decidedly enlarged. While the right tube and ovary are adherent, the ovary itself is apparently uninvolved, and the tube seems to open directly into the gestation sac. I take it that a rupture of the tube occurred between the folds of the broad ligament, that the impregnated ovum was not detached at the time, that hemorrhage was slight, and that the fetus continued to grow not only between the folds of the broad ligament, but lifted up the posterior layer of the peritoneum and continued its development subperitoneally. This form of ectopic pregnancy is what is known as the subperitoneo-pelvic variety, and should not be confused with secondary abdominal gestation, since the fetus, its membranes, etc., are distinctly and completely outside the peritoneal cavity.

What is the cause of ectopic gestation? That is a question of the greatest interest, in fact to me the most interesting feature in connection with the subject. While I know there are many who differ from me, and whose opinions I duly respect, and while I do not assert that my own view is correct, personally I think it is due to an atavistic tendency of the generative organs, if I may be permitted to use such a term; in other words, that we have here an evidence of evolution, that is, evolution of the generative organs from the bifed tubes, such as are seen in many of the lower mammals and the bifed uterus, such as is familiar to us in dogs and cats, to the single uterus such as observed in the human being. The solution of this question in my opinion hinges upon the point in the generative tract where impregnation of the ovum takes place. I believe in the human being, as well as in some other of the mammalia, impregnation of the ovum normally occurs only in the uterine cavity, that the ovum becomes enseminated in the place where it is intended to become

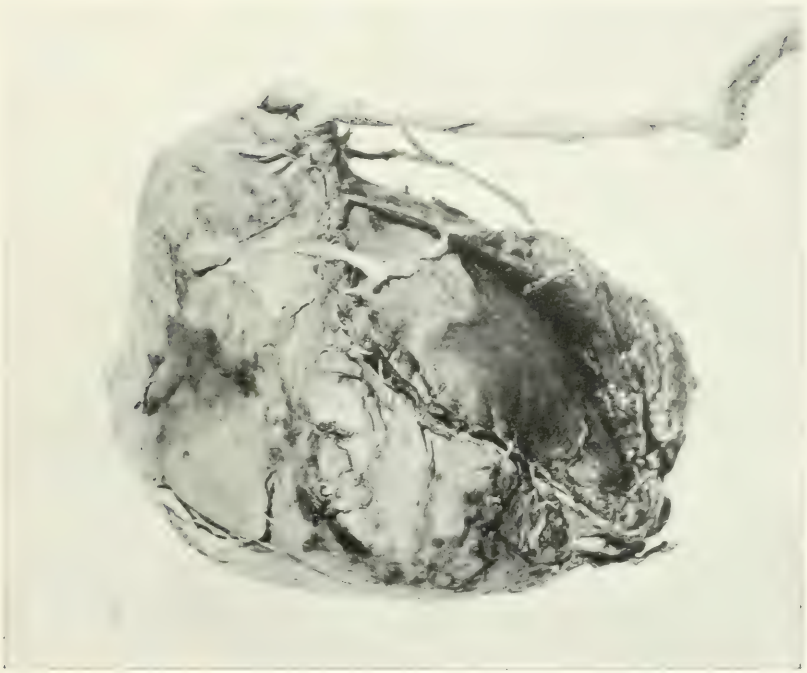

FIG. 1.—Specimen removed in Case I.

FIG. 2.—Fetus from Case I.

fixed and to continue its development, that when the spermatozoa ascend into the tubes and there impregnate the ovum we have cause sufficient for ectopic gestation. I contend that normally the ova are never impregnated in the Fallopian tubes, the teaching of physiologists and others to the contrary notwithstanding; that tubal disease, constriction by bands, pressure by tumors, and obstruction by anomalies of location have nothing whatsoever to do with the causation of ectopic gestation. I trust my meaning is clearly understood, but to recapitulate: normal pregnancy is impregnation of the ovum in the cavity of the uterus; impregnation in the tube is abnormal and always results, should the fetus continue to develop or become fixed, in ectopic gestation.

CASE II.—The second patient is also a negress, who was referred to me through the kindness of Doctor J. L. Atkinson, of Campbellsville, Kentucky. She gives a history which is much more indicative than the patient just operated upon, and there can be no reasonable doubt as to the condition present, the history being absolutely typical of ectopic gestation. By palpation we can distinctly outline the contour of the child, and there is not the least element of doubt in my mind as to the nature of the condition.

This woman is a little over 30 years of age, and is the mother of six children. In December, 1903, she had an abortion at the third month of utero-gestation. She menstruated normally in January and also in February, 1904; in fact, until July of that year menstruation was more or less regular. In July the flow lasted but a day or so, and the same was true in August and in September. These latter menstrual epochs were accompanied by pain, and the flow was not of the usual character. She says it was "splotchy;" that it would persist for a day or two in the form of small clots, and then cease. During October, 1904, she felt movements of the child and was much surprised thereat, though she had noted she was growing larger, but believed this was an ordinary increase in flesh. In October she did not menstruate at the usual time, but in the latter part of the month violent movements of the child were felt and copious menstruation occurred, after which no further fetal movements were observed, or, as she expressed it "everything became quiet again." She did not menstruate in November, but did so normally in December and again at the regular periods in January and in February

of this year (1905), the last menstruation being about two weeks ago. Between October, 1904, and the present time she has lost 50 pounds in weight, but she is still quite a large woman, as you observe.

Upon examination there is noted a tumor in the upper part of the abdomen extending transversely in its long axis from rib-line to rib-line. The growth is hard, nodular, and there is a distinct sulcus between it and the mass below, which examination shows clearly to be the uterus. Upon further investigation we detect what appears to be the head of the child on the left side, and a leg on the right side. There is apparently no communication between this tumor and the uterus. I hardly think there can be any question about the accuracy of our diagnosis, and the proper procedure is to make an incision and remove the fetus which, as in the other case, is now acting the part of a foreign body. There is still in this woman's breasts (as in the other woman's) some secretion.

Remember, this woman has lost flesh rapidly, and the clinical chemist tells us that there is a large percentage of sugar, as well as albumin, in the urine,—wherefore I shall complete the operation as quickly as possible. On making the incision we observe that the tumor is quite densely adherent to the anterior abdominal wall. I shall rapidly separate these adhesions, then open the sac and extract the fetus. Tracing the cord I find it extends to the right side, where I note the placenta is attached to the uterus deep down in the iliac fossa. On account of the vitiated condition of the woman, and the albumin and sugar in the urine, I believe it will be best not to subject her to a complete operation such as was performed on the other patient. I shall simply ligate the cord close to the placenta and remove the tumor mass, leaving the placenta, the gestation sac, the uterus and its adnexæ behind. The abdomen is closed with catgut by the usual tier method of suture without drainage.

It may not be the ideal plan of treatment to leave the placenta behind, but in this instance it is the proper thing to do. The child has been dead sufficiently long for the placental vessels to have closed, so there will be no danger of secondary hemorrhage, and, as she has had no evidence of infection thus far, we need not fear suppuration of the placenta; furthermore, just how long it might take to do a hysterectomy is unknown, and the patient's life might

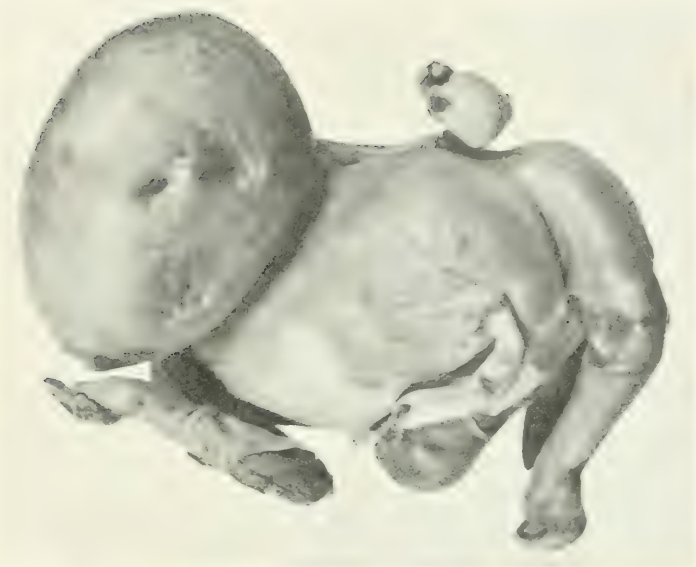

Fig. 3.—Fetus removed in Case II.

thereby be endangered. I do not believe the woman could withstand the prolonged surgical intervention to which the other patient was subjected, and remember, gentlemen, it is always the life of the patient that must be considered. Ideal surgery at times has to be sacrificed, and always should be sacrificed, if the patient's life is in the balance. It is better to save the life of your patient and not do such a beautiful operation, than to execute a more magnificent or brilliant surgical procedure and put the patient under the ground as a result! Surgery should always be life-saving. There is no absolute reason why the placenta in this case should be removed. Statistics show that patients do just as well when the placenta, in cases of this kind, is left behind as when it is taken away.

The fetus (Fig. 3) is still enclosed in the amniotic sac, which is closely adherent in various places; in fact, it seems to divide the fingers into two groups as it were; and the same condition obtains about the toes. Observe how closely the sac has drawn the elbows to the sides of the child. You might ask what has become of the amniotic fluid and the balance of the amniotic sac? The fluid has been absorbed, and the sac is continuous about the umbilical cord, as will be noted by careful inspection. This child's life seems to have been lost on account of compression of the cord by flexure of the leg and thigh. Probably during free movements of the fetus in the abdominal cavity, in which it lay enclosed in its amnion, the cord being attached below and there being an unusual range of mobility, it became entangled and thus perished. It had not gone on to full development, and, from the history of the case and the size of the fetus, I take it to be about a seven and a half months' child.

In this case the course of events has probably been as follows: The ovum became impregnated and implanted at the distal end of the tube, or it may be in one of the fimbriæ, and as it grew it gradually extended into the abdominal cavity, remaining enclosed in its amnion. The placenta, which at first found sufficient attachment to the fimbriated end of the tube, later formed other attachments to the posterior leaf of the broad ligament, where you observed it was firmly adherent, partially covering the uterus and also the upper part of the lateral pelvic wall.

In my opinion primary abdominal gestation is an impossibility,

and I believe it has been clearly proved that such never occurs; that it is always secondary to a tubal gestation; and this is an excellent illustration of the condition and one which to the inexperienced might appear as a primary abdominal pregnancy. All pregnancies, if not in the uterus, no matter whether in the broad ligament, in the retro-peritoneal pouch, or in the abdominal cavity, are always primarily and essentially tubal in character.

I believe both these patients have good chances of ultimate recovery. The surgery in the first case was quite extensive, and there may result some intestinal necrosis; there may even be a fecal fistula, but I think the patient will nevertheless recover. In the second case, provided there is no trouble from the kidneys—the presence of albumin or sugar in the circulation—I see no reason why the patient should not also get well. The only danger outside of diabetic or uremic complications which, had we been less careful, might have been a potent factor in the production of sepsis, is secondary hemorrhage from detachment of a thrombus at the maternal site of the placental attachment. Such an accident has occurred, but I do not anticipate anything of the kind in this case, on account of the time (about five months) intervening between the death of the fetus and the operation. The uterus will gradually become smaller, and it is possible that the woman may later bear children. Her child-bearing function has not been destroyed, and, while intrauterine development of a fetus might give rise to trouble, still should she become pregnant, it would be possible for her to bear a child in the normal way. We know that cases of simultaneous ectopic and intrauterine pregnancy have been reported. Nature sometimes does strange things, and this in itself is an evidence of some of her freaks.

[Both patients recovered completely.]

THE CHOICE OF MEDICINAL AGENTS FOR TOPICAL USE IN GYNECOLOGIC PRACTICE

BY CHAUNCEY D. PALMER, M.D.

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Gynecologic practice without doubt has been hindered in its progress by an unnecessary multiplication, and by certain combinations, of medical agents for local treatment of the diseases of the genital tract in women. That topical treatment is necessary for not a few of these local ailments is so apparent that any argumentation in its favor is uncalled for. Many of these affections are unrelieved without it, and not a few are cured in no other way.

Surgical operations are absolutely required in a large proportion of all cases, but medicinal applications only are needed in some cases and in others precede or follow the operative procedures.

Some of us can readily recall the influence of the teachings and writings of James Henry Bennett, of London, in his work on the Inflammations of the Uterus, published in several editions (1845-1861). This work was among the first in gynecology in the English language. None in medicine has ever exerted a more decided and profound influence in England, the Continent of Europe, or in America. We cannot forget the advocacy, by this author, of the use of silver nitrate, in solid caustic form, to the cervix uteri. All over the civilized world this and other potential caustics were used, and that too actively, for some supposed ulcerations of the womb. It was then deemed expedient and imperative to attack the disease at its supposed foundation head in this very way. This mistaken notion of the real pathology of many of the morbid conditions of the uterus—the idea that there was some peccant ulceration there located, which must be burned out, by some potential caustic, actuated the professional mind for many years. Ere long, fortunately,

it was found that as much harm as good resulted from such practices, and they were discontinued.

Very little need be mentioned as to the advisability and efficacy of the use of the mercuric bichloride, as one of the very best antiseptics and germicides we possess. It is constantly utilized now in some way for such purposes, preceding and following surgical operations in obstetrical and gynecologic work; it is uniformly beneficial, and very seldom indeed followed by any ill consequences. How frequently, too, may it be observed that the topical application of its solution (1 to 2000), following of course a thorough cleanliness of the parts, does enhance the efficaciousness of various other topical medicaments.

Nor need much be written concerning the large field of usefulness of boric acid, in saturated solution (22 grains to an ounce), as a mild emollient; and of the free application of boric-acid powder for similar purposes.

Lysol is another very useful disinfectant and antiseptic. Combined with soap, it makes a most excellent agent in the disinfection and the asepsis of the hands and instruments for intrapelvic examinations and operations. Lysol is from three to five times more powerful as a bactericide than is carbolic acid, and is much less poisonous.

Iodine has been employed for many years and in many combinations as a topical medicament in gynecologic practice. I have long used it in the following combinations, which is sometimes spoken of as Churchill's tincture:

℞	Iodine	2 drams	8.0
	Potassium iodide	4 drams	16.0
	Glycerin	1 dram	4.0
	Alcohol, sufficient to make	1 ounce	30.0

Mix. Sig. To be used locally for cervical and intra-uterine purposes.

Dr. Robert Battey, of Rome, Georgia, being unfavorably impressed with the results obtained from intrauterine medication with silver nitrate and nitric acid, instituted a series of experiments to find an eligible substitute—one that would be efficient as a local remedy, and at the same time leave the uterus in a normal state. He found that for ordinary purposes, the solution of two parts of iodine, to eight parts of carbolic acid, proved to be the best. This preparation was named by him as iodized phenol.

All written in praise of this remedy by Battey has been substantiated by many gynecologists. It is a comparatively painless medicament, thoughly antiseptic, and very valuable. It is one of the best known topical medicaments for intra-uterine purposes. As a rule it is my choice of remedies for local medication, after the minor operation of uterine dilatation and packing. The formula I have used is:

Iodine	100 grains	6.0
Carbolic acid	1 ounce	30.0

An excellent addition to the list of gynecic remedies was made when ichthyol was introduced to our notice. This, the sulpho-ichthyate of ammonium has a decided antiseptic action, being actively fatal to pathogenic organisms; is well tolerated when applied to the endometrium, inasmuch as it reduces swelling and relieves pain in inflammatory states of its surfaces. We can then, assert with confidence, that it is antiseptic, analgesic, and antiphlogistic. Erosions and granular degenerations, and chronic follicular ulcerations, so called, of the cervix uteri are almost always quite favorably modified by its local use; so, also, are conditions of old, long standing inflammations of the bladder, with or without papillomatous formations, as well as chronic inflammations and ulcerations, and fissures of the rectum. For intra-uterine medication, seldom indeed need aught else be utilized than ichthyol or iodized phenol. I feel confirmed in the utilization of ichthyol incorporated with glycerite of alum or of boroglycerite, applied by wool tampon, for the relief of many cases of chronic peri-uterine inflammations. What ichthyol is to the dermatologist, a valued remedy, as much or more it is to the gynecologist.

Reference has been made to the use, the advantages and the disadvantages, of the topical employment of silver nitrate, in solution and in crayon. Its application within recent years has been so much superseded by various so-called organic preparations of silver that are equally efficacious, less painful, and not followed by any sangineous discharge, or by the least persistent contraction of the cervical canal of the uterus, so that fortunately the older forms of silver may be entirely discarded. Because of the amount of silver contained in these preparations, their solubility in water, and because they do not coagulate albumen, nor precipitate the

chlorides, and because they do not produce any irritation or pain when applied, or destroy tissue, they are of signal service in the treatment of certain diseases of the genito-urinary system of both sexes.

Many of the most active chemical caustics, as nitric acid, the acid nitrate of mercury and caustic potash, may, in our present therapeutic light, be dismissed from further gynecic use. Of them there is nothing superior to the zinc chloride in from 25 to 50 per cent. solutions, for the destruction of malignant neoplasms. When carcinoma of the uterus has extended beyond the limits of a thorough and complete eradication, an abatement of the local ravages and an amelioration of the offensive discharges and hemorrhages may be obtained by an active sharp curettement of the whole endometrial cavity; thus, after the arrest of all of the flow of blood therefrom, a free application of this salt may be made with benefit. Of course the vaginal canal should be properly protected from this liquid caustic, and some anodyne, for the relief of local pain, will probably be needed; but the local conditions are much better thereby; so life may be much prolonged. Experience has strengthened my faith in the efficacy of this zinc chloride solution, as a most serviceable caustic to the uterus and its cavity, in not a few cases of cancer of this organ, when the time for a complete hysterectomy has passed, the condition in which so many patients first come under the physician's observation and care.

Chromic acid, 50 per cent., is at times a convenient and beneficial caustic for gynecic use. Slow in its action, deeply penetrating in its effects, not particularly painful, it has for itself a limited field of usefulness.

A great change for the better in the management of chronic uterine affections has been secured by the abandonment of many of the most active chemical caustics, and the substitution instead of less painful but more remedial local medicaments.

Bismuth salicylate is a most valuable topical medicament for chronic inflammatory conditions of the vagina and cervix uteri. Thus, after the most thorough sharp curettage of the uterine neck and cavity for cancer, in cases in which the uterus is fixed, and beyond the time for a complete abdominal or vaginal extirpation, a very useful local medication is the free application of this powder

every few days, throughout the entire curetted tract. Should some remedy more active be called for, choice may be made of a powder of salicylic acid and chlorate of potassium, each equal parts.

Adrenalin has been found to be a medicinal substance of more than ordinary power as an astringent and hemostatic; hence its inestimable value in controlling losses of blood in various minor surgical operations on the vulva, the urethra, the vagina, and uterus. Its power in this direction makes it one of the most serviceable agents ever discovered in the realms of medicine. The pallor of the conjunctiva induced by its instillation into the eye, the checking of persistent hemorrhage following gum lancing and teeth extraction, are evidences of what may be expected on the female genital tract under similar circumstances.

For instance, the complete and thorough exsection of irritable caruncles from the female urethra is of no infrequent necessity. A long-continued oozing of blood from the urethra when these growths are high in this canal is at times very troublesome and annoying. A free application of this solution (1 to 2000) serves under such circumstances a most useful purpose.

No topical medicament for gynecologic use has been utilized more frequently in the last decade or two than has the glycerite of boroglycerin. The best preparation should be made 50 per cent. strong and of pure anhydrous glycerin, applied incorporated into pure absorbent cotton, then placed against the cervix, or into the anterior or posterior vaginal cul-de-sacs, as local conditions call for; and largely as an emollient, antiseptic, and antiphlogistic. But a superior local medicament for the rectification of conditions of uterine displacement, associated with and usually dependent upon changes in the circulation, if not some chronic inflammatory lesions of the uterine tissues, is a combination of equal parts of this glycerite of boroglycerin and the glycerite of alum, slightly thymolated, and incorporated into tampons of absorbent wool instead of cotton.

Other agents might be named, but my personal experience prompts me to refer to the above-mentioned as those most needed in almost all instances and conditions of gynecologic practice.

Laryngology

INTRATRACHEAL AND INTRALARYNGEAL INJECTIONS

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Many of those who have given thorough trial to intratracheal and intralaryngeal injections have written enthusiastically in praise of these therapeutic procedures. They declare that patients are relieved of distressing symptoms and improved in general health as by no other method, and they make it evident that these injections have a distinct field of usefulness.

Why the injections are so seldom employed is understood when one considers that patients must be treated every other day or even daily, and that the technic cannot be acquired without some care and patience; besides, certain persons with very sensitive throats are difficult to treat, the number getting smaller as the physician's technic improves.

My own belief is that anyone attempting to use these injections as a routine practice will be disappointed in them; on the other hand, if the physician reserves this method for patients with distressing symptoms that have failed to receive benefit from the usual modes of treatment, he will often be surprised and pleased by the readiness with which he can give the intralaryngeal injections and by the beneficial results that follow.

History.—Early in the nineteenth century, twenty years before Garcia perfected the laryngoscopic mirror, attempts were made to inject medicines through the larynx, though it is doubtful whether

the trachea was reached by the method employed. In 1837, Trousseau and Belloc [1, 2] recommended a syringe with a long curved cannula for injecting a spray into the larynx, and in 1847 Green [3, 7, 8] expressed very positive views concerning the practicability of pulmonary medication by means of a long, flexible tracheal tube; he used silver nitrate in 10 to 20 per cent. solutions. Green met with much opposition from the medical profession. A partial bibliography of the historical side of the subject will be found at the end of the paper.

Diseases for Which the Injections are Employed.—In general it may be stated that intralaryngeal and intratracheal injections are of use in chronic cases when the mucous membrane of the air tract is, (*a*) too dry, (*b*) covered with tenacious secretion, (*c*) broken by ulceration, or (*d*) has on its surface virulent microorganisms. They are especially serviceable when it is necessary to leave the stomach undisturbed. According to the medicine used, the injections will act as a sedative, an expectorant, or a germicide.

The following list includes the diseases in which good may be expected from this method of treatment: Chronic catarrhal cough; chronic tracheitis; asthma; chronic bronchitis; bronchiectasis; bronchorrhea; abscess of the lung; gangrene of the lung; hemoptysis [19] (often a contraindication); tuberculosis of the lungs, and chronic laryngitis, whether simple, tuberculous, or syphilitic. Sometimes intratracheal injections act as a specific in the chronic affections named in the above list—in asthma, in the dry cough of beginning tuberculosis of the lung,[21] and in chronic laryngitis, especially when associated with lithemia.[8] In diseases of the air tract with tenacious, abundant, or fetid secretion and discharge, such as chronic bronchitis, bronchiectasis,[21] bronchorrhea,[30] advanced tuberculosis of the lung, and abscess and gangrene of lung, great benefit is afforded by this treatment, relief from cough, pain, and expectoration being experienced in necessarily fatal cases.[19, 21] In ulceration of the larynx or trachea, whether tuberculous or syphilitic,[21, 29] healing is hastened and pain relieved.

Some observers claim notable results in advanced tuberculosis of the lung from the use of antiseptics by intratracheal injection, believing that cavities are reached and disinfected.[19]

Contraindications.—Intratracheal injections do no good and may do harm if used in the acute hyperemic stage of any type of inflammation.[8] Hemoptysis is also usually considered a contraindication.

Anatomic and Physiologic Factors.—Barton[24] states that the average trachea is about four and a half inches long and from three-quarters to one inch wide, and he estimates that the lung alveoli expose an absorbing surface of 130 square meters. Experiments on dogs[9] seem to prove that oily liquids can pass into the bronchi without causing injury and be absorbed into the circulation. Olive oil is rapidly taken up by the lymphatics and can be detected in the parenchyma of the lung soon after the intratracheal injection has been given, deep inhalations carrying the volatilized vapor deep into the bronchioles; alkaloids promptly manifest their physiologic effects and iodine can be detected in the urine. Patients state they can smell and taste guaiacol six to twelve hours after treatment, and it is sometimes possible to detect the odor of the medicine on the breath for twelve hours.[10] The treatment, however, is employed for its local, rather than for its systemic, effect—to allay irritation, promote free secretion of seromucus, and prevent fermentation or decomposition of secretion.[8]

Dangers.—Reflex spasm of the glottis,[31] pneumonia,[32] and pulmonary hemorrhage may be induced by the injection of fluids into the trachea through the larynx. Such complications and sequels are not common. Spasm of the glottis is not likely to occur to any serious extent, local anesthetics being employed to prevent it.[33]

Medicaments.—The following vehicles are popular in the order named: Pure olive oil;* commercially pure petroleum oils,† such as benzoinol, liquid albolene; Price's redistilled glycerin; and water.

Sedatives.—Menthol, 1 to 10 per cent.; heroin, $^1/_{24}$ grain (0.0025 gram); morphin, $^1/_8$ grain (0.008 gram); orthoform, 12 per cent.; anesthesin, 2 to 6 per cent.; chloroform, 5 minims (0.30

*Olive oil has a tendency to corrode metal syringes, and may become rancid.

†Petroleum oil may be tested by dissolving iodine crystals in it; if the iodine is not reduced and remains in solution unchanged, as may be proved by the starch test the oil is pure.

c.c.); compound tincture of benzoin, 5 minims (0.30 c.c.); chloretone, 1 per cent.; and camphor, 0.5 to 2 per cent.

Expectorants.—Creasote, 1 to 2 per cent.; guaiacol, 1 to 2 per cent.; iodine, sufficient to make a pink solution.

Antiseptics.—Oil of thyme, 2 per cent.; oil of eucalyptus, 2 per cent.; oil of cinnamon, 2 per cent.; oil of gualtheria, 2 per cent.; salol, 3 per cent.; ichthyol, 2 per cent.; iodoform, 2 per cent. ($\frac{1}{8}$ grain); and potassium permanganate, ($\frac{1}{8}$ grain). The amount per dose is given in parenthesis.

The mixture that I have used most is made up as follows:

℞	Creasote	5 to 10 grains	0.30 to	0.60
	Menthol	5 to 15 grains	0.30 to	1.00
	Olive oil	1 ounce		30.00

Mix. S. Two drams (7.5 c. c.) to be injected at a dose, daily, every other day, or twice weekly.

To each dose of the foregoing the following may be added: heroin, $\frac{1}{24}$ grain (0.0025 gram); morphin, $\frac{1}{8}$ grain (0.008 gram), if cough is a prominent symptom; or iodoform, $\frac{1}{2}$ grain (0.03 gram), if expectoration is fetid or contains tubercle bacilli.

It is well to begin with 1 per cent. menthol and rapidly increase the strength. Guaiacol may be substituted for creasote, and other combinations of drugs may be made according to the symptoms present in the individual patient. The following formulas have been much commended:

Mendel's [34]

℞	Iodoform			
	Guaiacol			
	Essence of thyme			
	Essence of cinnamon			
	Essence of eucalyptus	of each	5	00
	Olive oil		100	00

Mix.

For tuberculosis Campbell [19] uses during the first week of treatment:

℞	Thymol			
	Menthol	of each	1 dram	4.00
	Salol		2 drams	8.00
	Glycerin (Price's redistilled)		5 ounces	150.00

Mix in a 6 ounce (180 c.c.) bottle. Dissolve on a water bath.

Sig. Two to twelve drams (7.5 to 45 c.c.) to be injected twice daily.

During the second week of treatment and later, Campbell uses the following:

℞	Medicinal izal	3 drams	12|00
	Menthol	2 drams	8|00
	Glycerin (Price's redistilled)	5 ounces	150|00

Mix. Sig. Two drams (7.5 c.c.) to be injected once daily.

For tuberculous ulceration of the larynx with painful deglutition, the following are recommended:

℞	Heroin hydrochlorate	3 grains	0|20
	Water	1 ounce	30|00

Mix. Sig. A few drops of a warmed solution to be applied by means of a syringe to the ulcer before meals.

Freudenthal's emulsion consists of the following:

℞	Menthol	1.00	5.00	10.00	15|00
	Orthoform				12|50
	Oil of sweet almonds				30|00
	Egg yolk				25|00
	Distilled water, sufficient to make				100|00

Mix and make an emulsion.

Sig. A few drops to two drams (7.5 c.c.) to be used as a dose.

It is well to begin with 1 per cent. menthol, and quickly increase to 10 per cent. Menthol and orthoform in liquid albolene may be used instead of the emulsion.

Another formula useful in odynphagia is:

℞	Menthol	1|5	22½ grains
	Powdered gum arabic		
	Oil of sweet almonds		
	Distilled water, of each	10|00	2½ drams

Mix; make an emulsion; and add

Anesthesin	1 to 4|00	15 to 60 grains
Brandy	20|00	5 drams
Distilled water	60|00	2 ounces

Mix. Sig. Two drams to be injected into the larynx. "No intoxication need be feared, and anesthesia lasts from 3 to 30 hours."

Syringe.—The Muir syringe (Fig. 1.) has long been a favorite. The wide barrel can be replaced by a narrower one (Fig. 2). The syringe has a flexible curved cannula about seven inches long, and holds about 4 fluid drams (15 c.c.). Donnellan [15] employs a glass syringe with a metal cannula bent at a right angle at about two inches from the end. The tip is perforated at the side

(Fig. 3) so that the solution is not projected directly down the trachea. Hubbard [8] uses a cannula with a half-inch bend, not long enough to reach below the tip of the epiglottis. Others depend upon an atomizer with a down tip and with it spray into the larynx for from 30 to 60 seconds.

Anesthetic.—Although in many instances intratracheal injections can be made without local anesthesia, it is sometimes necessary

Fig. 1.—Muir syringe.

to employ it, reflex spasm being lessened thereby. Some physicians use a 4 per cent. cocain hydrochlorate solution on a cotton-tipped laryngeal applicator, touching under full illumination first the pharyngeal wall and the epiglottis, and afterward the lower structures of the larynx down to the vocal cords, a period of five minutes being then allowed for the drug to take effect. Others [8] use 0.5 to 1 per cent. cocain solution, which is sprayed into the larynx.

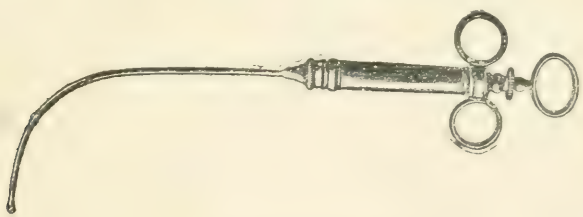

Fig. 2.—Syringe used by the author.

Goodale [32] uses a saturated solution of cocain-β with a few drops of adrenalin chloride, 1 to 1,000. Roe [32] allows the patient to inhale a few drops of chloroform until slight effects are felt, in this way sensibility is lessened and other anesthetic is not required.

Technic.—It is well to begin treatment by cleansing the nose and larynx by spraying or applying a non-irritating alkaline liquid such as normal salt or boric-acid solution, after which the throat

is anesthetized by one of the methods given above. A period of five minutes is allowed for the anesthetic to take effect, while the syringe and bottle of solution to be used are warming in a basin of water at a temperature of about 100° F.

There are two ways of giving the injections: (1) Without the laryngoscope—"going it blind"; and (2) with the mirror.

Without the laryngoscopic mirror. The tongue is protruded and held by the left hand of the physician, and the injection of 1 to 2 ounces (30 to 60 c. c.) of the medicament is made while the patient holds the breath, or during deep inspiration the tip of the syringe can be directed against the side of the pharynx. Of course, the injection causes coughing, but scarcely as much as one might expect, and enough solution will be inhaled into the trachea to bring about therapeutic results.

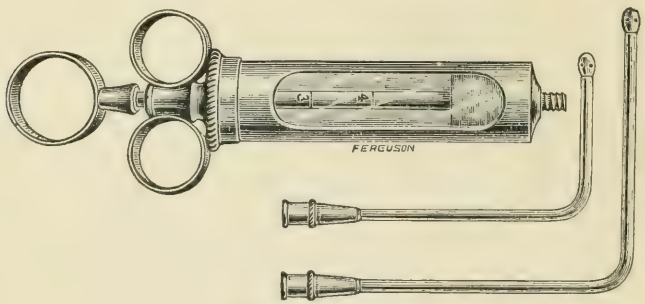

FIG. 3.—Dounelian syringe.

With the laryngoscopic mirror. A preliminary few days' training of the throat is of value in gaining the patient's confidence and in establishing a tolerance for the introduction of instruments. When the injection is to be made the patient protrudes his tongue and holds it with his right hand, while the physician places the warmed laryngoscopic mirror against the soft palate and guides the syringe under full illumination, without touching the pharyngeal wall, base of the tongue, or the epiglottis, into the larynx, and, if desired, on between the vocal cords. The injection is made slowly while the patient holds his breath or during deep inspiration, 1 to 2 drams (4 to 8 c.c.) being injected. (Figs. 4, 5, 6, 7.)

If the cannula with a half-inch bend is employed, the tip is held just beyond the epiglottis and the syringe discharged during

short, quick breathing. Hubbard [12] tried this method on himself and found it entirely satisfactory.

Typical Cases.—The following detailed histories are fairly representative of the results in favorable cases by intratracheal injections:

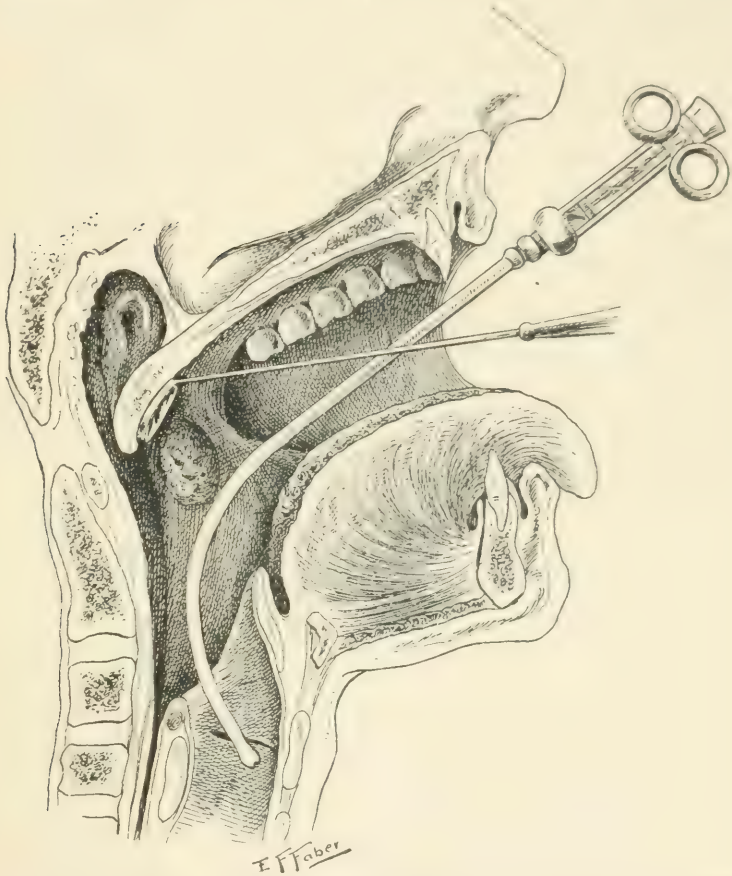

Fig. 4.—Diagram representing a median section, to show the laryngoscopic mirror and cannula of the syringe in position.

Case I.—A. K. W., an American of Irish parentage, aged 18 years, a seamstress, had had rheumatism, measles, whooping cough, and mumps when young. In April, 1905, she contracted a severe cold from which the attack of tuberculosis seems to date. She was treated at a sanitarium for a few weeks, and later came to the physician who referred her to me. At the first examination very

few lung symptoms were found; when seen some months later, however, distinct evidences of diffuse disease of the left upper lobe were evident, together with some pleuritic involvement.

When seen by me, early in December, 1905, she was suffering from cough, muco-purulent expectoration, hoarseness, and pain on swallowing of a severe type, radiating to the left ear. After a few days training daily injections were given without difficulty: menthol 5 grains, creasote 5 grains, and liquid albolene 1 ounce, being used at first; later menthol 10 grains, creasote 5 grains, iodoform 15 grains, and olive oil 1 ounce; and still later menthol 10 grains,

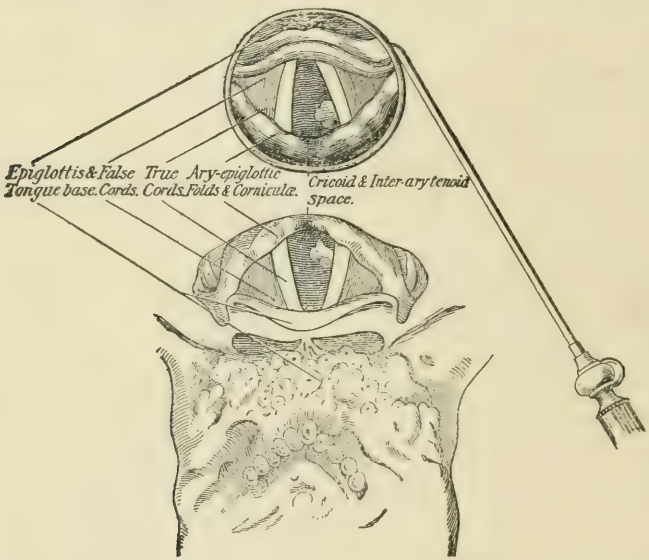

FIG. 5.—Diagrammatic view of tongue-base, epiglottis, arytenoids and aryepiglottic folds, ventricular bands, and vocal cords, with the laryngoscopic reflection. A polyp shows below the left cord. (Schrötter.)

creasote 5 grains, europhen 20 grains, and olive oil 1 ounce. At the beginning one dram was injected daily; this was increased rapidly to three drams. On two occasions pure lactic acid was applied to the larynx, followed by the oil solution and orthoform powder. The laryngeal lesions were of a serious type—infiltration and ulceration of the ventricular bands and interarytenoid fold, and some subglottic swelling.

The patient steadily improved from the first. At the end of two weeks pain on swallowing had practically disappeared, though it returned when the injections were omitted for a few days. It

FIG. 6.—First step. The patient protrudes the tongue and holds it with [illegible] hand, while the physician places the mirror against the soft palate and holds it up.

FIG. 7.—Second step. The syringe is introduced into the larynx in full view, without touching the tongue, pharynx, or epiglottis.

again disappeared after three days, and after a week injections were given every other day instead of daily. Later the interval was increased to twice a week. At present, January 23, 1906, she is in very good condition: She has no pain on swallowing, her cough is less, the expectoration has nearly lost its purulent character and in every way her improvement has been satisfactory. The larynx has changed for the better to a considerable degree.

Case II.—G. K., an Irishman, 49 years old, a weaver by trade, was referred to the medical dispensary of the University Hospital for asthma in October, 1905. He had used alcohol in moderation, and tobacco to excess. Since an attack of pleurisy 14 years ago he has had cough, dyspnea, and yellowish expectoration; has lost strength and weight the last two months. He appears cyanotic, his chest is emphysematous, with poor expansion, hyperresonant everywhere except at the right apex where it is impaired; wheezing breath sounds, expiration prolonged. No edema. The heart sounds are weak, the heart is slightly enlarged. No tubercle bacilli are in sputum; the urine is free from sugar and albumin. Pulse 96; temp. 99.4°.

Examination of upper air tract showed general chronic congestion; nasal fossæ free, some swelling of the arvepiglottic folds.

The patient was placed upon potassium iodide 10 grains (0.6 gram) three times a day, and heroin $\frac{1}{8}$ grain (0.008 gram) four times a day. Later tincture of belladonna 5 minims (0.30 c.c.) was given for a short time. Under this treatment the patient would improve for a week or two and then relapse to his previous state. In January of this year (1906) intralaryngeal injections of creasote 5 grains, menthol 5 grains, and liquid albolene 1 ounce, 2 drams at a time, were given every two or three days with marked beneficial results; all symptoms—cough, dyspnea, and expectoration—being greatly lessened. The patient declared after ten days of treatment that he was able to sleep better than he had for many months, and could not be made to believe that his imagination had anything to do with it.

Summary.—Intratracheal and intralaryngeal injections are often found of value in *chronic* affections of the mucosa of the larynx, trachea and bronchi *after other* methods of treatment have failed. A warm solution can in many cases be injected into the

larynx with or without the aid of the laryngoscopic mirror, and be expected to produce a sedative, expectorant and antiseptic action without disordering the stomach. The injections should be made deliberately into the partly anesthetized larynx during deep inspiration, while the breath is held, or while the patient is taking short, quick breaths. Injections should not be employed when the mucosa is acutely inflamed.

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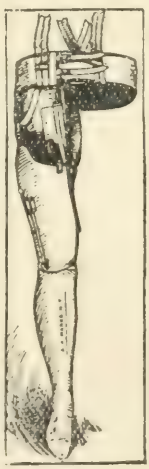

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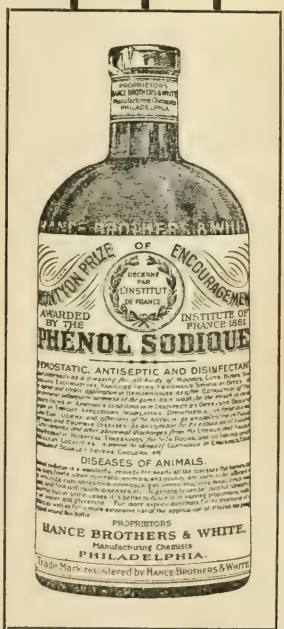

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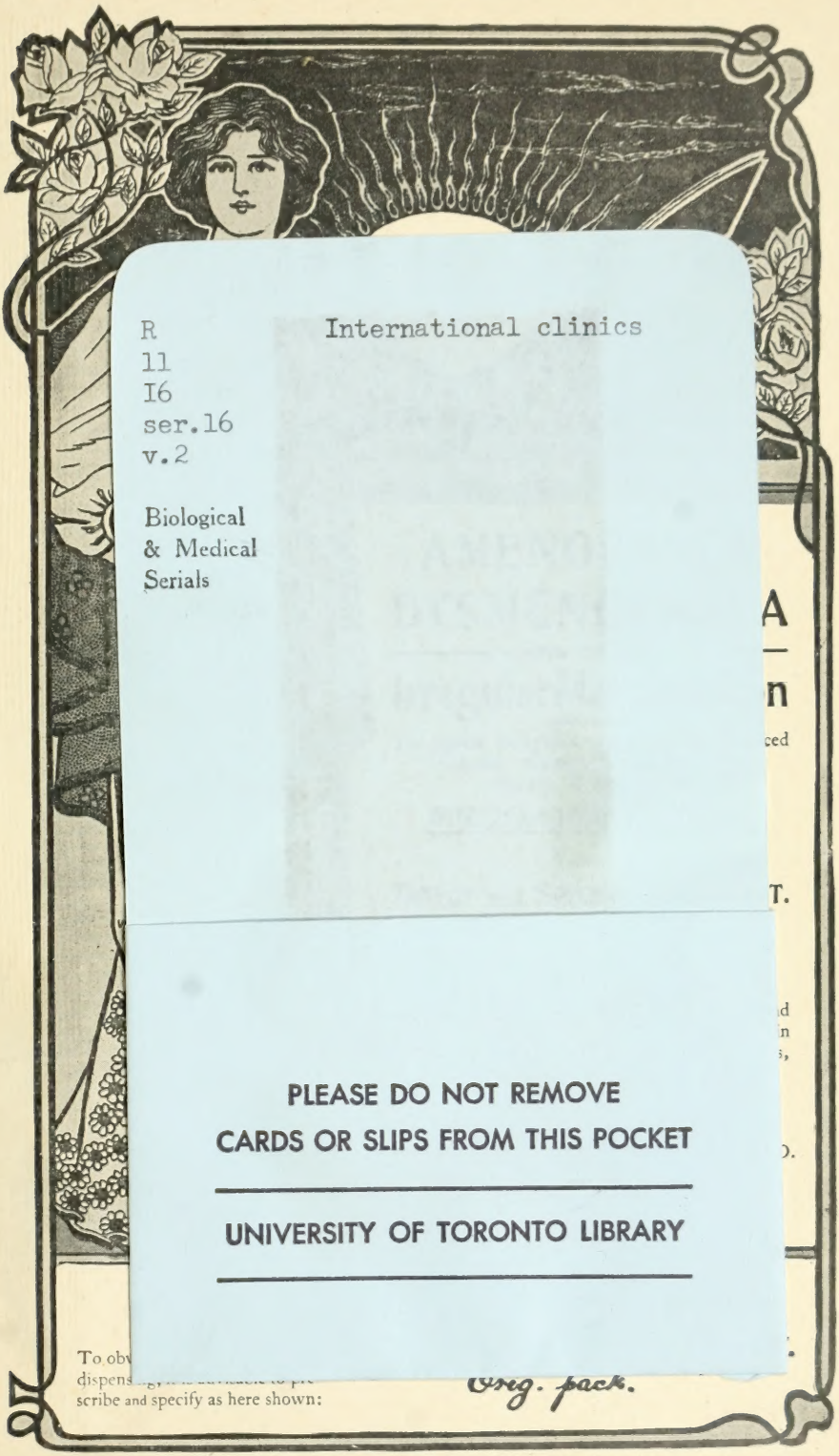

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